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Pathological Changes in the Peridental Membrane.—*Noyes.*

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Phenomena of Inflammation.—*McConnell.*

Tooth Brush and Methods of Cleaning the Teeth.—*Sarrazin.*

Fundamentals of Periodontology.—*McCall.*

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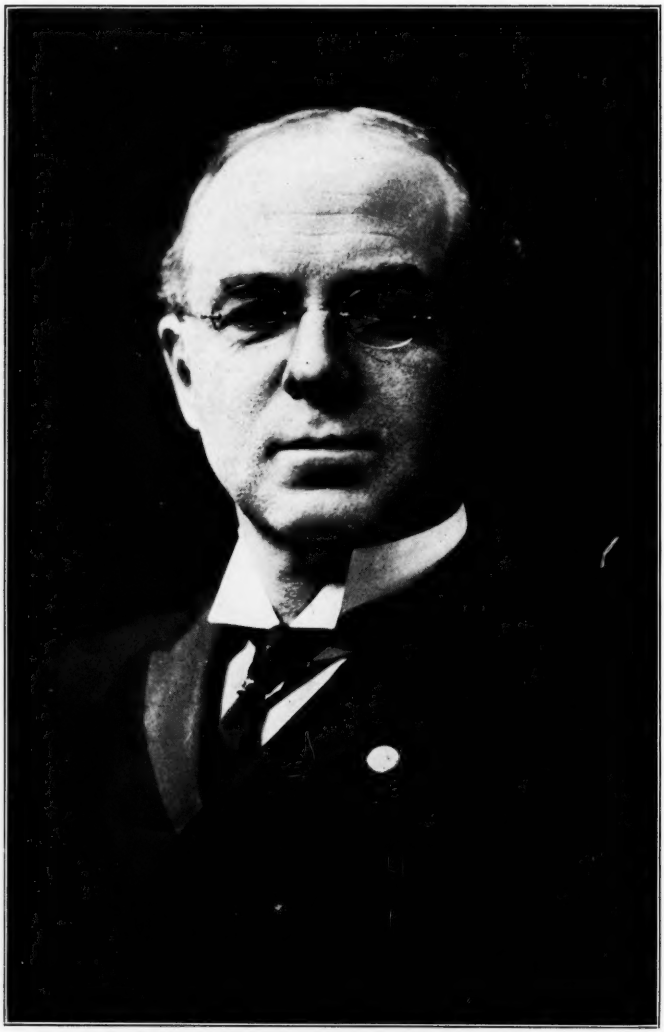

DR. CHARLES SIMEON BUTLER.

THE JOURNAL
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Original Communications.

PATHOLOGICAL CHANGES IN THE PERIDENTAL MEMBRANE.

By Frederick B. Noyes, D. D. S., Chicago, Illinois.

(Read before the Illinois State Dental Society, Peoria, May 13-16, 1919.)

FOR several years most of the work in my laboratory in the Dental Department of the University of Illinois has been devoted to the study of the structural changes in the peridental membrane and the supporting tissues of the teeth. This work was begun with the assistance of the Research Institute of the National Dental Association. The work done for the institute has never been published, because when it was ready for publication, my grip, containing all of the material was stolen. The grip contained not only the manuscript and the carbon of it, but the laboratory note book with the notes of more than two years' of work, the illustrations, including twelve or more water color drawings that had cost over two hundred dollars, more than one hundred slides selected from the different series, including all of the best ones and the ones from which the illustrations had been made, in short everything except the negatives that had been made from the sections. The war has made the repetition of this work for publication impossible. Part of what is here given is the result of that work.

The study of a large volume of ma-

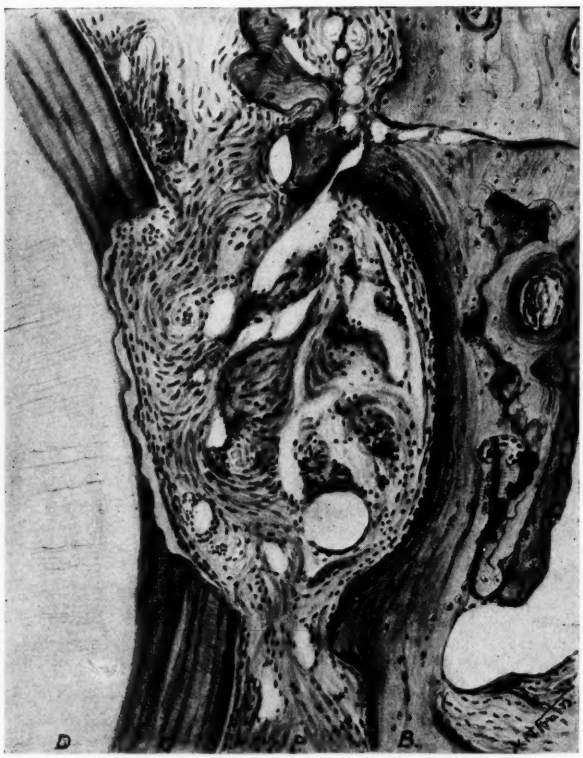
terial has given me the impression that there may be in these destructive inflammations several more or less distinct pathologic processes, which have more or less distinct characteristics, but to sort out the material so as to build up the pathological picture for each will require a vast amount of work.

Gradually however two or three things have taken on sufficiently distinct form to admit of clear statement. These I am going to present to you. They may be summed up under three heads.

(1) The destructive inflammations of the peridental membrane are caused by recurrent infections producing intermittent, progressive destruction of the characteristic arrangement of the tissues.

(2) The course followed by the destructive inflammation is determined by the penetration of the infection along the course of the lymphatics.

Figure I.



Transverse section thru the tooth and alveolar process showing area of destructive Inflammation.

B, Bone. C, Cementum. D, Dentin. P, Peridental membrane.

(3) Two distinct types may be recognized and related to the source of the infection and the course of the lymphatics along which it progresses.

Figure II.

Transverse section thru the tooth and alveolar process showing area of destructive Inflammation.

B, Bone. C, Cementum. D, Dentin. P, Peridental membrane.

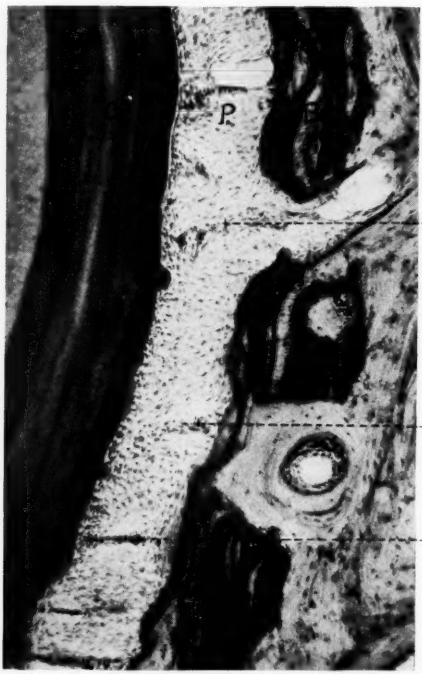
I. *Recurrent infections causing intermittent progressive destruction of characteristic arrangement.*

For many years the men who have studied most carefully the changes in the tissues occurring in the inflammations of the peridental membrane have been convinced that the conditions found were the reaction of the tissue to recurrent infections. In the study of the present material one of the first things to take clear form was the record in the tissues themselves of the intermittent and progressive character of the disease. Figures I and II are from transverse sections of the tooth and alveolar process showing marked inflammatory reaction.

The illustrations reproduced as ac-

curately as possible the conditions shown in the sections, but the staining in the specimens shows by differences in color of bone and cementum of different periods of formation more clearly than is possible in a monochrome. Careful study of these pictures will show that there have been successive periods of acute inflammation, with destruction of the principal fibers of the peridental membrane, bone, and cementum, followed by periods of quiet or chronic condition. In each period of quiet there has been partial restoration with new formation of bone and cementum, only to be followed by a new acute infection with added destruction. The different shadings indicate the different formations, and the irregular lines show where a new formation began to be laid down on the surface where an absorption had stopped.

Figure III.



Photomicrograph of a transverse section showing nearly normal peridental membrane.

I. Interfibrous tissue.

By cutting deeper and deeper into the tissues, that is farther and farther from the gingiva where the infection began, it was sought to discover the first deviation from normal appearance in the tissue. After studying many sections, from a large number of teeth, it became apparent that the first positive changes

occurred in the interfibrous tissue. That is, in the slight amount of connective tissue that normally fills the spaces between the principal fibres which pass in bundles from the cementum to the bone. Normally this tissue is small in amount, and surrounds and accompanies the blood vessels.

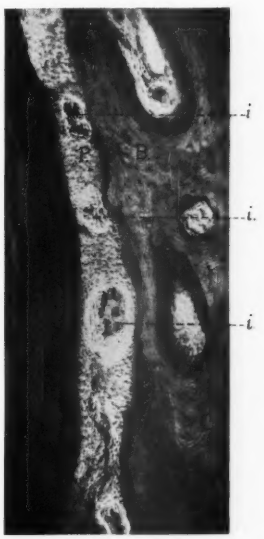
Figure III is taken from deep in the tissues beyond the point showing any distinctly pathologic change. The peridental membrane is approximately normal in thickness and character. It will be seen that the principal fibres constitute almost the entire tissue, the slight amount of interfibrous tissue is indicated at the points lettered I. If this picture is compared with figures IV and V it will be seen that the difference is in the increased amount of this interfibrous tissue and the inflammatory character of it. These spaces between the principal fibres become filled with inflammatory products which encroach more and more on the principal fibres, pushing them aside. The blood vessel walls in these areas also show inflammatory reaction. By the continuation of this process the normal arrangement of the tissue becomes profoundly altered. Figures VI and VII show the way in which this progresses. The two photographs are taken from the same tooth at different levels. Figure VI being nearer the gingiva than figure VII and therefore in a more advanced stage. In figure VII it will be seen that the inflammatory tissue has greatly increased in the areas of interfibrous tissue, and has pushed the principal fibres aside. In

Figure IV.

Photomicrograph of a transverse section showing slight inflammation of interfibrous tissue.

i. Showing increased amount of interfibrous tissue displacing the principal fibers.

Figure V.



Photomicrograph of a transverse section showing slight inflammation of interfibrous tissue.

i. Showing increased amount of interfibrous tissue displacing the principal fibers.

several places it has encroached upon the bone, producing absorption. The principal fibres, compressed between these inflammatory areas, show marked degeneration in the faint staining of their fibroblasts, and a loss of distinctness of tained to the cementum. This results in a large area of inflammatory tissue which destroys the normal structure of the peridental membrane and encroaches upon the bone of the alveolar wall. The normal thickness of the membrane is

Figure VI.

Transverse section showing breaking of principal fibers,—fusion of centers of inflammation,—destruction of bone and large area of inflammatory tissue.

C, Cementum. B, Bone. P, Peridental membrane.
PF, Ends of principal fibers still attached to cementum.
i. Interfibrous areas fused into large mass of inflammatory tissue.

the individual fibres and a change in their character. They tend to take on a fused hyaline appearance. In figure VI it will be seen that the areas indicated by I in figure VII have united, breaking the principal fibres and destroying the bone to which they were attached but leaving their degenerated ends still at- still shown at the upper and lower edges of the picture.

From a study of these sections it became clear that infections begin at the gingival margin, and at least in some conditions there are fingerlike extentions of inflammatory tissue stretching deeper and deeper into the tissues, and

it became more and more apparent that there was some determining factor in the course which the infection follows. It did not seem probable that this was simply the formation of the interfibrous spaces nor that the infection followed

lymphatics of the peridental membrane was undertaken.

II. *The relation of the lymphatics to the extention of the infection.*

Dr. Kaethe W. Dewey and the writer conducted a series of injections to ex-

Figure VII.

Transverse section a little deeper than VI, showing the extensions of inflammatory tissue in the interfibrous tissue.

I, Interfibrous tissue. C, Cementum. B, Bone. P, Peridental membrane.

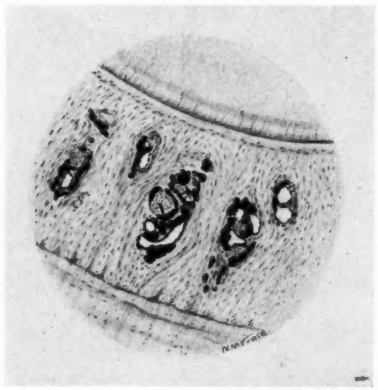
the walls of the blood vessels, and the character of the reaction in the blood vessel walls seemed to indicate that it was exterior to them rather than being carried in the blood current. The most reasonable hypothesis seemed to be that the infection progressed along lymphatic vessels, or lymph courses. To furnish light on this an investigation of the

amine the lymphatics of the peridental membrane. This work was published in the Journal of The American Medical Association, October 12, 1918, Vol. 71, P. 1179, 1184. The findings may be summarized in a few words. The work of Sappey had shown that the lymphatics of the gum pass over the buccal and lingual surfaces of the alveolar pro-

cesses, outside of the periosteum to form a wreath-like network where the tissues are reflected from the bone to the cheek or tongue. These are drained by a few efferent vessels whose course we need not follow here. Sappy did not show any lymphatic vessels in the peridental membrane.

Figure VIII shows injected lymphatic vessels in the peridental membrane, and by comparison with the previous illustrations it will be noted that they are found in exactly the positions in which the first inflammatory reactions occur.

Figure VIII.



Drawing from transverse section showing injected lymphatic vessels in the peridental membrane of a dog.

Figure XIV is a longitudinal section showing lymphatic vessels injected and extending from the connective papillae under the epithelium lining the gingival space, penetrating the ligamentum circulara, and extending into the peridental membrane. Comparing with the work of Sappey, therefore, it appears that while the lymphatics from the papillae of the labial and lingual slopes of the gingiva pass outside of the periosteum and over the labial and lingual surfaces of the bone of the alveolar process, the lymphatics from the papillae of the gingival space penetrate the ligamentum circulara and extend into the peridental membrane.

Figure IX.

Drawing of longitudinal section, showing injected lymphatic vessels.

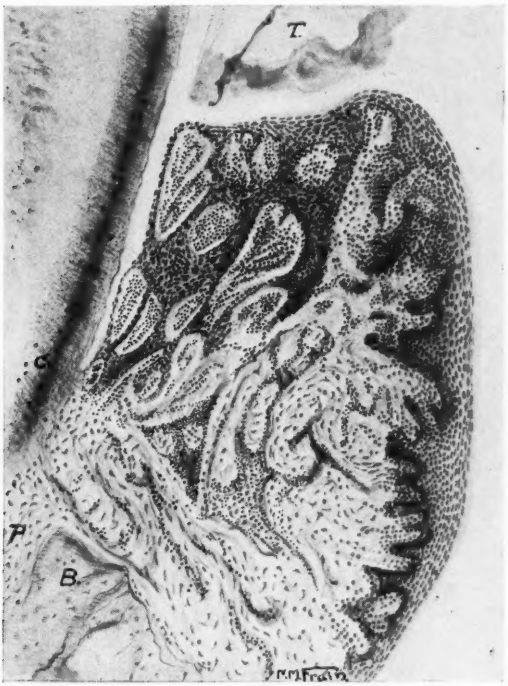
G, Crest of gingiva. Gs, Gingival space. C, Cementum. D, Dentin. B, Bone at crest of the alveolar process.

III. *Two distinct types of tissue change.*

Before the influence of lymphatics in the extention of the infection was suspected, it became apparent from the study of material from different cases

that there were two distinct types of tissue change, illustrated in figures X and XI. In figure X there is shown at T a mass of salivary calculus which has broadened and flattened at the over the lingual (or labial) side under the periosteum, but on the peridental side there is new formation of bone and the fibres of the peridental membrane show no destruction or degeneration.

Figure X.



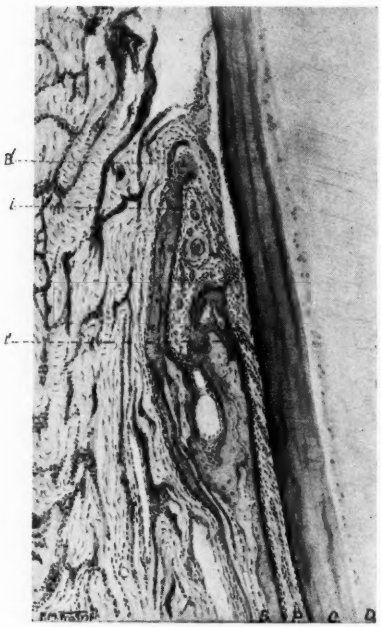
Drawing of a longitudinal section of gingiva.
T, Salivary calculus. B, Bone. P, Peridental membrane.

gingiva. From this broad, flat area infection is entering into the tissue producing inflammatory reactions in it. Dr. Hatton has been able to stain the microorganisms in such tissue and so demonstrate their presence. (Journal of the American Medical Association, 1918.) The bone of the alveolar process is shown at B and it will be noted that it shows absorption over the tip and extending Sections that were lost show the conditions much more clearly than this, because of being cut in exactly the right position and direction.

Figure XI is also a longitudinal section of the tooth and supporting tissues showing marked inflammatory reaction, but in this case the infection was apparently from within the gingival space. The bone forming the tip of the

alveolar process is indicated at B and it will be seen that there has been destruction of bone on the side of the peridental membrane and new formation of bone under the periosteum causing the border of the alveolar process to become thickened and turned outward.

Figure XI.



Drawing of a longitudinal section showing penetration of inflammation into the peridental membrane.

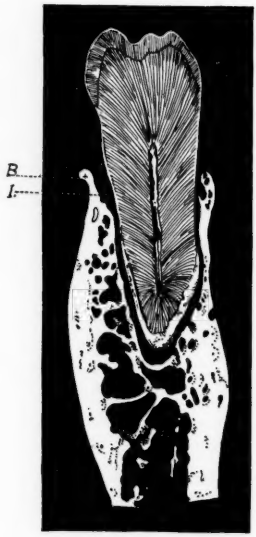
B, Bone. C, Cementum. D, Dentin. P, Peridental membrane. B', Bone at the border of the alveolar process. i, Inflammatory extension of interfibrous tissue corresponding to Figure VI. I', Area corresponding to Figure VII.

The characteristic structural arrangement of the peridental membrane is destroyed in the region indicated by I, and in its place there is found a much larger amount of inflammatory tissue. The area indicated by I corresponds to the position shown in transverse section in VI and the area shown at I corresponds with the level shown in figure

Figure XII.

Mandible showing eversion of the border of the alveolar process around the molars.

Figure XIII.



Diagramatic drawing showing eversion of the border of the alveolar process, which is filled with inflammatory tissue.

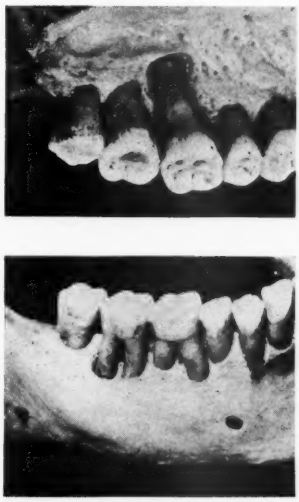
VII. At I there has been much more destruction of bone and the characteristic structure of the peridental membrane is almost entirely gone, but two-thirds of the way towards the bottom of the picture, where the thickness of the peridental membrane is still about normal, the tissue shows increased interfibrous spaces filled with inflammatory tissue, and the structure and staining of the bone shows that there have been periods of acute inflammation and resolution.

XII is a drawing of a mandible which shows this condition around the molars. Note the thickened, dense, everted border of the alveolar process. This leaves a space next to the tooth which in longitudinal section is V shaped and is filled with inflammatory tissue. Figure XIII was made as a diagram of the condition. In these cases the infection seems to have followed the lymphatics into the peridental membrane. Figure XIV shows photographs from another skull, showing the same conditions.

In the condition shown in figure X the infection has apparently followed the lymphatics over the outer surface of the alveolar process. The destructive inflammation occurs under the periosteum and the peridental membrane remains comparatively normal in structure.

It has not been the intention to present this as a report of investigation as to those trained in such work. The mass of material is still too confused for such presentation, but out of this mass of material, probably representing more than one pathologic picture, these few

Figure XIV.



Photograph of upper and lower molars showing destruction of bone and eversion of the bony border.

things have taken form sufficiently clear to be expressed. The tissue conditions clearly correspond to clinically recognizable conditions, and it is hoped that the description of tissue conditions may help in guiding, to some extent at least, the treatment of the tissues and the prevention of recurrent infections.

TREATMENT OF CHRONIC SUPPURATIVE PERICEMENTITIS.

By Arthur D. Black, A.M., M.D., D.D.S., F.A.C.S., Chicago, Illinois.

(Read before the Illinois State Dental Society, Peoria, May 13-16, 1919.)

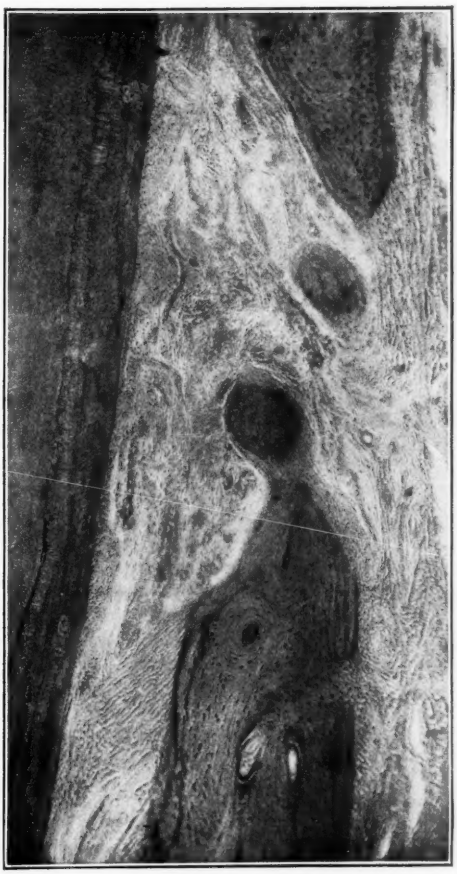
THE term chronic suppurative pericementitis is applied to that disease of the investing tissues of the teeth in which a pus pocket is formed along the side of the root. The pathological changes presented in the paper by Dr. F. B. Noyes are of two distinct conditions; the one caused by deposits of salivary calculus, in which the infection is described as traveling along those lymphatics which are attached to the vessels of the gum tissue, *on the outer side of the bone of the alveolar process;* the other resulting from an initial gingivitis (caused in the main by injury, or deposits of serumal calculus) which travels along the lymphatics attached to the vessels that course thru the peridental membrane, *on the inner side of the alveolar process.* Clinical observation supports these laboratory studies, and both lead to the conclusion that deposits of salivary calculus are not to be considered a cause of the formation of pus pockets.

Before taking up the treatment of pus pockets, one or two additional statements regarding the pathological changes should be made. In the summer of 1912, I suggested to my father, who was then preparing the manuscript for his book on Special Dental Pathology, that we should examine microscopically the tissues overlying the teeth, to see what changes had taken place as a result of the infection and pocket formation. Soon afterwards, I began cutting away sections of tissue for this purpose, occasionally removing parts of the alveolar process, peridental membrane and overlying gum tissue with teeth which were condemned for extraction. We soon accumulated many specimens which showed the changes which Dr. Noyes has described in detail. One specimen in particular, Figure I, shows practically all the changes except the actual formation of the pocket. The fringe of short ends of fibres attached to the cementum is very clearly shown.

After there is an actual break in the tissue, which apparently occurs about midway between the surface of the root and the bone, the tissue attached to the cementum is exposed to the fluids of the mouth, and it soon dies, leaving the cementum denuded. At the same time, the vitality of the cementum is lost as a result of the destruction of the cementoblasts, which lie upon its surface. It is comparable to a piece of necrosed bone which can not be exfoliated, and remains as a constant irritant to the adjacent soft tissue. Upon these pathological changes, the treatment which I will present, is based. It should be recognized that practically all hope of reconstruction of the peridental tissues to normal, or of reattachment to the cementum is gone forever.

The pathological picture of these overlying tissues is one of recurrent infection, which agrees with clinical observation of cases in practice. A pocket once formed is therefore a menace to health in that it offers opportunity for the accumulation of organisms in contact with a connective tissue thru which

Figure I.



Section thru root and labial alveolar process of an upper central incisor, showing about one-fourth of the root close to the apex. Patient forty years of age. The peridental membrane on the lingual side of this tooth was detached almost to the apex and the pocket extended around on both the mesial and distal sides of the root, but was not so deep on either the mesial or distal, as on the lingual. One band of good peridental membrane fibers is shown near the bottom of the illustration; higher up (towards the gingiva) much degeneration of the peridental membrane has taken place, altho many bundles of short fibers remain attached to the cementum. The general trend of the blood vessels is apparent. This illustrates the destructive changes which occur within the peridental membrane in advance of the actual break or formation of a pocket.

there is easy access to the circulation. To treat these pockets in what seems to be the most rational way today requires that one recognize clearly the meaning of the pathological changes which have been presented. If this is done, then we will give up the idea so long held that our effort in treatment is to bring about a reattachment of the overlying tissue to the root. Having done this, three principal methods of operative procedure may be recommended.

Figure II.

Normal peridental membrane. Section showing fibers attached to the cementum from the gingival line almost to the apex of the root. The section is not quite parallel with the long axis of the tooth and is a little to one side of the apex of the root. The fibers which pass upward into the gingiva, those which pass to the crest of the alveolar process, and those which pass directly from the cementum to the bone are clearly shown as a practically solid mass from the gingival line to the end of the root. A blood vessel is shown arching over the crest of the alveolar process, others may be seen within the peridental membrane, their course being parallel to the long axis of the root.

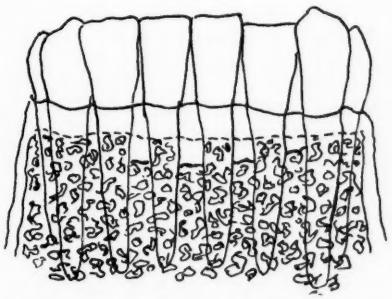
First, the extraction of those teeth about which we may not hope to prevent reinfection by other methods of treatment. The deeper the pocket, the greater the indication for extraction. The position of the pocket must be taken into consideration; for example, a proximal surface pocket of a given depth will indicate extraction, when a pocket of the same depth on a labial or buccal surface might be treated satisfactorily by other means. Much looseness of the tooth, during a period when there is little or no inflammation, would suggest the advisability of removal. Failure to prevent reinfection by other methods of treatment calls for extraction. After a thoro examination, those teeth which are marked for extraction, should be removed at the earliest opportunity.

Second, for those teeth which have pockets, but which are not to be extracted, our problem is that of preventing reinfection. The first procedure is to eliminate irritation, both by the removal of deposits and also by the correction of fillings, crowns, etc., which directly as by bad margins, or indirectly as by bad contacts, cause inflammation. When all sources of irritation are removed, the soft tissue will usually rid itself of infection if the pocket is thoroly cleansed by irrigation with a mild antiseptic, or with physiological salt solution. Then the problem arises of preventing reinfection of the pocket. To do this, cleanliness of the pocket must be maintained. In the majority of cases this can not be accomplished by the dentist alone, unless the patient is required to return at very frequent intervals, and even then there is

an occasional reinfection in most cases with gradual progress of the pocket in depth.

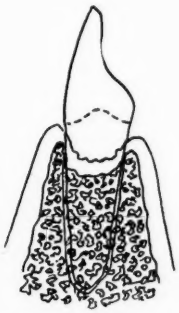
It is strongly urged that in all these cases the patient be taught to use a rubber bulb syringe with a large nozzle, and instructed to wash the pockets out with warm salt water twice daily, as a part of the routine technic in mouth hygiene. In cases in which this cooperation by the patient is secured, reinfection of pockets is effectively prevented and they may go indefinitely without further progress or systemic danger.

Figure III.



Sketch of the lower front teeth, labial view. There are interproximal pockets between the incisors, also between the left lateral incisor and cuspid. A line indicates the position of the crest of the gingiva. The broken line indicates the crest of the bone. The depth to which the interproximal bone has been destroyed also is shown.

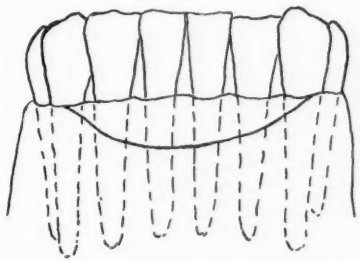
Figure IV.



Sketch of one of the lower incisors shown in Figure III, proximal view. The interproximal pocket, with the destruction of the central portion of the bone, is shown, with the labial and lingual portions of the process about normal height. To eliminate such a pocket it is clearly necessary to cut away the gum tissue and bone on the labial and lingual at least to the level of the interproximal bone.

Third. In many cases in which there are pockets, by far the simplest and most effective method of treatment is to cut away the overlying tissue and thus eliminate the pocket. When this can be done, no further treatment is required by the dentist and no special care is necessary on the part of the patient. To carry out this procedure, a rather definite technic is necessary, based upon the conditions presenting. We may divide cases into three groups: those in which there are pockets on labial or buccal sides of the roots, those in which there are proximal pockets, and those in which there are pockets which encircle teeth.

Figure V.



First incision in operation to eliminate pockets. This is a curved line from cuspid to cuspid, the intention being to remove all of the soft tissue and bone to the incisal of this incision. Novocain is injected and the incision made with a small straight knife on the labial side. A similar incision is made on the lingual side with a knife having a blade set at an angle of about forty-five degrees to the handle.

The operation is performed under novocain anesthesia. The effort is made in each case to remove all overlying tissue, thus laying the root bare to the depth of the pocket. To do this, it is usually necessary to trim off a little of the crest of the process because the detachment of the peridental membrane from the cementum is in advance of the

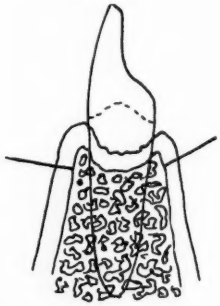
destruction of the bone. This can be observed in radiographs as well as in the microscopical specimens. It is also important that the bone be cut to a slightly deeper level than the gum, in order that the cut edge of the gum may slightly overlap the edge of the process; this promotes rapid healing and prevents necrosis of the margin of the bone.

In cases in which there are pockets on labial or buccal surfaces, an incision is first made thru the gum on a line about even with the edge of the bone; that is, the line to which the bone has been destroyed, removing all soft tissue crownwise of this line. No interproximal tissue is removed if there are no proximal pockets. Next the edge of the process is trimmed off with an excavating chisel or a fissure bur the end blades of which have been ground off so that the end may be held against the root, while the side blades cut the process. By either plan the gum tissue is pressed aside and immediately afterwards will overlap the cut edge of the bone. The area is thoroly irrigated with hot saline solution until the hemorrhage has ceased, when the patient is dismissed with instructions to rinse the mouth thoroly with warm salt water after each meal until the gum has healed so that the tooth brush may be used. Occasionally, if much of the cementum is thus exposed, the teeth will be slightly sensitive to thermal changes for a week or two. We have had but one case in which the sensitiveness required attention; it was relieved at once by the application of silver nitrate crystals.

In operating on cases of this group, it is desirable that the line of the incision be a gradual curve; there should be no sharp angles. If there is a single deep pocket, as on the labial surface of one lower incisor, it will be of little benefit to cut off only the detached tissue, because on account of the narrowness of the notch, the tissue from the sides would grow across, reproducing the pocket. It is sometimes necessary to cut away a little tissue from a neighboring tooth in order to give a more even curve to the gum margin after healing.

The elimination of labial pockets is often contraindicated because of the unsightliness of the denuded roots, particularly in the upper incisor region. Similar pockets on lingual surfaces of the

Figure VI.



Proximal view, showing position of labial and lingual incisions in relation to depth of interproximal pockets.

Figure VII.

Proximal view showing large chisel (20-9-6 Black's formula) with cutting edge placed in previously-made incision. Chisel is forced thru bone to root; this is repeated several times for each tooth until all of the bone is cut thru. Then with a scraping motion in the incisal direction, the bone and soft tissue are removed. This is repeated for each tooth. Sometimes the same chisel may be used on the lingual, or an instrument having a greater angle of blade may be more convenient.

lower teeth may be treated in the same way. Pockets on the lingual of the upper teeth are less often benefited by this operation because of the thickness of the bone as it curves away to form the palate, which leaves a broad shelf next to the tooth after healing.

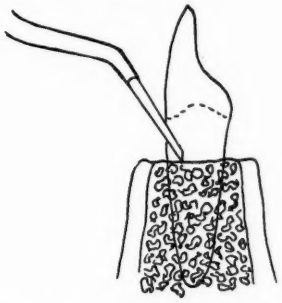
In cases in which there are proximal pockets, the operation is more difficult, as it is necessary to cut away the buccal and lingual bone and gum opposite the interproximal space, even with or a little farther apically than the depth of the interproximal pocket. This must be done in order to eliminate the pocket, so that, after healing, the patient may take a mouthful of water and by forcing it back and forth between the teeth, wash the interproximal regions thoroly. Most such cases are due to lack of contact or improper contact of the teeth, and it is of course important that a proper contact be restored.

In cases in which the pockets encircle the teeth, the operation is very simple, consisting of the removal of the unattached soft tissue and a little of the edge of the alveolar process. This condition most often presents in the mouths of persons who have been immune to decay, and have therefore neglected to care for their teeth by proper brushing or by sufficiently frequent visits to the dentist for examination and prophylactic treatment. There is likely to be a more or less complete ring of deposit of serumal calculus encircling each tooth, beneath the gum margin. This has resulted in the infection of all of the gingiva, and eventually in the formation of pockets of about equal depth encircling all of the teeth.

If the investing tissues about all of the teeth are involved, I have usually divided the operation, cutting away the upper and lower gum on one side at the first operation, and the upper and lower on the other side at a later time. Or sometimes one side of each arch has been operated separately, making four operations in all.

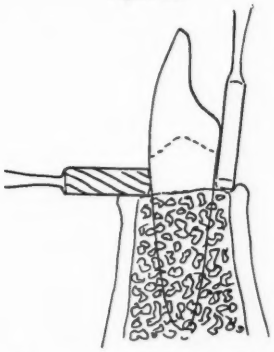
This plan of eliminating the pockets has greatly simplified the management of those cases which were most difficult and unsatisfactory as previously treated. The operation is completed in a very few minutes, and no further treatment is necessary. Many cases have

Figure VIII.



A chisel of suitable size is carried thru each interproximal space to remove any unhealthy granulation tissue which may remain in the central portion. Usually a little of the crest of the bone is cut away.

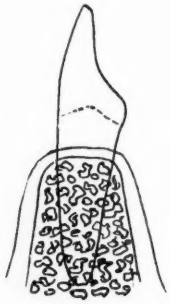
Figure IX.



This illustrates two methods of trimming the crest of the bone on the labial and lingual sides of the teeth. This bone should be cut a little shorter than the proximal bone; it should be trimmed sufficiently to permit the cut edge of gum tissue to overlap a little. This facilitates the building of new epithelium over the bone. A fissure bur, used as indicated, should have a non-cutting end. The end blades of an ordinary fissure bur may be ground off.

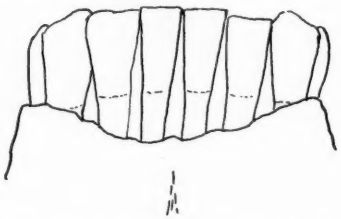
been kept under observation for several years without requiring further attention. Teeth, which have been very loose, have become much more firm and have remained so. Patients generally express gratification at being free from the intermittent attacks of sore teeth which are

Figure X.



The proximal view about two weeks after the operation, showing growth of soft tissue covering the bone.

Figure XI.



Labial view of case after operation. All pockets have been eliminated. The wide open interproximal spaces may be kept clean by rinsing the mouth thoroly with water after each meal.

symptomatic of recurrent infections. Many teeth which would otherwise be extracted to remove the menace of systemic infection, may be safely retained. We have used this method extensively during the past seven years and our results have generally been satisfactory; it has come to be our routine method of treatment for the majority of cases presenting.

Discussion.

E. H. Hatton, Chicago.

These papers are most important because they deal with fundamental principles of the anatomy and pathology of the teeth and adjacent structures. No system of technical procedure or treatment can endure unless it is based upon these principles. Because of this the demonstration of the presence and distribution of lymph channels in the peridental membrane and pulp is a matter of the highest importance. Infection elsewhere follows lymph channels, and the study of diseased peridental membrane and pulp argues for the same course here. Indeed it has been shown repeatedly that the advance of the infection does parallel the course of the lymphatics as demonstrated by Noyes and Dewey; their preparations are most convincing and a distinct advance beyond any previous work, indeed it is still taught that there are no lymphatics in the pulp.

All of the gum tissue removed by Dr. Black in his clinic has been sectioned, and it is possible to say that the changes seen are uniform and that they are largely perivascular in location. It is possible to show that there is very little unchanged gum tissue in these specimens, and that the operation is conducted so as to conserve as much as possible of the structures. The operation is simple, and yet technically well planned. The results are excellent and lasting beyond reasonable expectation.

In applying these papers to practical affairs there are two statements to be made. First, the dental and medical professions are united in the belief that the teeth and the surrounding structures offer locations for foci of infection, and that everything is being done to protect the rest of the body from the results of such infection by a rather radical revision of both medical and dental practice. Second: The dental

profession must not overlook the conservation of the teeth and mouth structures as far as it is reasonably safe. From a decade of criminal conservation of diseased teeth, the practice has swung to the equally undesirable and serious sacrifice of blameless teeth.

Such papers as the two under discussion furnish a basis for harmonizing and correlating these two matters, and nothing is more important now in the practice of dentistry.

F. W. Merrifield, Chicago.

Mr. President, Members of the Illinois State Dental Society, Ladies and Gentlemen:

In our considerations of this subject we might well divide our attention between some of predisposing causes of peridental infection and destruction, and the different means by which these conditions may be remedied or cured.

Perhaps one of the foremost in the first division is malocclusion:

Broadly, malocclusion may be considered as of two types.

1. That in which the teeth are in abnormal occlusion from infancy and secondly that type which is induced by faulty dental operations.

These latter, in which contacts are not restored—where normal spacing is not obtained and where fillings, etc., are so placed that occlusion, both when the teeth meet their antagonists in direct closing and during masticatory effort—is not properly restored. In both of these types trauma to a greater or lesser degree is present—leading to a weakening of the resistance of the soft tissues and opening avenues for a possible infection.

In the first type, the orthodontist can and is doing much to prevent peridental destruction. In cases of the second type the general practitioner must assume much responsibility.

Teeth are removed with little or no thought for the care of spaces created and the consequent opening of contacts and the excursion of teeth thru the arches. Fillings are left too high, bridges are made which do not allow of normal mastication and which eventually destroy themselves. Many other factors will be found and while little dental restoration is perfect, there is room for much more care and consideration of these conditions.

Taking predisposing conditions from another angle we find injuries caused by some operative and prosthetic procedures. Fillings placed without regard for the normal space mesio-distally will result in the crowding of gum tissue in the interproximal areas with the consequent irritation, injury, infection, and perhaps destruction.

It will be well to mention factors which originate with the individual patient. The excessive and unwarranted use of toothpicks, the over-zealous use of dental floss and of the tooth brush, may each produce serious results, altho perhaps very much less harm is done with the brush in its over-use than its neglect.

Attention to the foregoing will result in much prevention which means more than cure, but what of the cases we are called upon to cure? A chronic suppurative condition of the peridental membrane, may be considered as one of the three classes:

First, that in which the removal of deposits, polishing these surfaces and the correction of fillings, etc., may effect the cure, almost without exception.

Second, that in which the suppurative process has progressed to the point where a considerable area of the peridental membrane attachment is destroyed with the resulting formation of a pocket, but with a not too great involvement of this structure or the alveolus, and

Third, that in which almost all or the greater part of the attachment is destroyed with the accompanying alveolar absorption. In cases of this type extraction appears to be the logically sanitary treatment.

Cases of the second class are the ones which are claiming the attention of the profession the world over as keenly now as ever. In our treatment of this type of cases, we must approach from several different angles. We must know the tissues involved, their functions, histology and their pathology—we shall then know what we cannot do or expect and then we have a reason upon which to base our hopes of success.

Another angle, and an important one, that of cooperation on the part of the patient. I believe that a great percentage of failures arise from lack of real interest and cooperation given by the patient, either along the line of general care and cleanliness or perhaps in regard to suggestions offered as to diet.

Still another angle—that of expediency. Do patients appreciate long drawn out, extensive scaling, planing and polishing operations and subsequent reporting at frequent intervals over a future indefinite period and then only so that the suppurative process may be controlled and not eliminated? When we realize that a detachment of the peridental membrane as the result of a suppurative process involves not only the death of the cementum of the root itself but also the destruction of the cementoblasts in the membrane which provide its attachment to the cementum—then only can we appreciate the futility of attempting to cure these conditions by any means which permits the overlying tissue forming the pocket to remain rather than to take the obvious and easy—tho radical method of removing it surgically.

During the last two or three years I have had opportunity to watch the results in cases treated in such a manner, that is, eliminating the pocket formations by cutting entirely away and followed with a good measure of success. Such a method recommends itself for the rapidity with which infection may be eliminated and the condition improved.

While all cases so treated are not uniformly successful, it is more likely to prove successful in restoring health to the tissues involved.

Dr. Collins: I would like to ask Dr. Black whether, when he referred to contact, he meant any space at all existing between the teeth or only where retention of food will apply. We know in many mouths in which there are spaces between the bicuspids, the tissue is perfectly healthy. If that is the case, would it not be advisable in some of these patients to grind away and polish some of the proximal surfaces below the contact point, so that food will not stay?

I would also like to ask him, if the pocket is shallow and the deposits are removed, whether the gum at that point does not atrophy, and what about removing the gum tissue over the pocket without cutting it away?

Q. How does this surgical operation that Dr. Black describes affect the teeth? Does it make them extremely sensitive and annoying to the patient, or is there any danger of thermal changes causing death of these pulps?

Q. When the teeth are scaled, are they scaled before operating or at the time of operation?

Dr. Black (closing the discussion): I will confine my remarks to replying to the questions that have been raised. First, in regard to the possibility of gum tissue receding over a shallow pocket, and if there is anything more done than to scale or clean that area. That happens in a good many cases and is a condition in which nature accomplishes just the same thing that I am proposing we should do when nature fails to do it. It does not happen in all cases by any means, because in a great many cases, even with a shallow pocket, we will have a swelling of that tissue and of the gingiva. In these cases I will cut the tissue away, but in the majority of cases where the pockets are quite shallow it is unnecessary to do

this operation. I would not have you think for a minute I would suggest the application of this operation to all cases in which we have pockets, because a great many of them can be better treated as we have been accustomed to treat them.

About the question of contact. We all, I presume, recognize that there are a great many cases where there is a wide open space between the incisor teeth in which no food will collect because the space is so wide, and these tissues remain in a healthy condition. We less often see that between bicuspids and molars where the force used in mastication is heavier. I would not grind off the surfaces of the teeth to make a more open space so that food would not wedge. We do not have the same conditions we have between the incisor teeth where the spaces are wide open. The normal contour of the proximal surfaces directs the food slightly away from the tooth surface where the food comes in contact with the gingiva. If we grind off the convexity, the food strikes on the edge of the gingiva where it meets the tooth and it is more likely to be inflamed. If the teeth are so close that food catches, let us remember that they are usually close enough so that we can build out the contact. I prefer to cut a cavity in a sound tooth and put in a filling to build out the contact rather than grind the surfaces away. There is an occasional case in which I have done what has been suggested; I have cut away and opened the contact wider.

As to the sensitiveness of these teeth, where very much of the cementum is exposed, such as occurs in these operations, where we cut the gums away from five or six teeth, these teeth will be sensitive to the extremes of heat or cold for a week or two. We have had but a single case out of several hundred that have been operated on in my clinic in which reaction to thermal shock required treatment. We went over the surfaces of the cementum with silver nitrate and the sensitiveness was relieved at once. The teeth were comfortable after that.

In answer to the other question, I would scale these teeth after cutting the tissue away because it is much easier to do. On the other hand, there are a limited number of cases in which the tissues are badly inflammed as the case presents and in which it is desirable to remove the bulk of the deposit about the teeth and wash the pockets out with normal salt solution over periods a few days apart to reduce the inflammation before we cut away the tissue, because we will have so much hemorrhage and may be less successful in obtaining anesthesia with novocain while there is so much inflammation. It is a good principle in surgery to reduce very marked inflammation before you inject and cut away this tissue.

We have a group of cases almost by themselves in which the pockets are about the lingual roots of the upper first molars. If these are very deep and involve the bifurcation, I would, as a rule, prefer to amputate the lingual root of the tooth, because in the majority of cases, particularly in the upper first molars, if you have destruction of bone and changes around the root into the bifurcation, there is no way of securing a reasonable amount of cleanliness. With lower molars it may not be so necessary to amputate the roots. I have seen occasionally a case of a lower first molar in which we have cut away bone and the overlying tissue below the bifurcation and epithelium was built over the cut surface thru the bifurcation, leaving it the same as in the case of an old person in which the bone and the overlying soft tissues have receded half the way down, the roots being exposed at the bifurcation of the teeth, and yet there is no inflammation.

THE PHENOMENA OF INFLAMMATION—A PRACTICAL INTERPRETATION.

By Guthrie McConnell, M. D.

From the Research Institute of the National Dental Association.

(Read before the National Dental Association at Its Twenty-third Annual Session, New Orleans, La., October 20-24, 1919.)

BEFORE beginning a detailed description of the development of inflammation it should constantly be kept in mind that this process is a protective reaction of irritated and damaged tissues which still retain vitality. If the tissues are dead they are unable to give rise to any reaction, it is the still living cells in the neighborhood that are able to respond to stimuli and that can carry on the activities necessary to repair, either by regeneration or replacement, the damage that has been done.

In taking up the study of the manifestations of disease in the human body, it must be remembered that we are dealing with biological processes of a very complicated nature. Altho they are abnormal as compared with the conditions existing in the healthy individual yet, in their fundamental activities, they are primarily physiological. They are governed and controlled by the same regulating mechanisms as exist in health. Those changes that when so grouped as to constitute a definite entity called a disease are made up of normal materials reacting to abnormal influences. We are apt to forget, for instance, that the changes occurring in chronic heart disease are but the logical outcome of the complicated forces acting upon that organ. In a case of aortic obstruction it becomes necessary for the heart muscle to become larger and stronger in order to force a sufficient supply of blood thru the vessels to supply the demands of the tissue. In the course of time the heart becomes unable to carry on the extra work and the symptoms of heart failure make their appearance. Under the microscope a sarcoma and a mass of granulation tissue may appear almost identical. This can be understood when it is realized that the cells in the two formations have the same origin. The difference, clinically, between the two is that in the tumor the cells never grow up, they never mature. They seem to possess to an exaggerated degree the ability to increase in number, but they have lost their power of specialization, and consequently they no longer serve a useful purpose. On the other hand, in granulation tissue, the cells are all the time under a controlling influence that permits them to grow to a certain extent, then calls a halt upon their activities. When the damage has been repaired no more cells are required, multiplication ceases, certain retrogressive changes take place and the process is over.

In the days of the early investigators, the various diseases and lesions that

were observed were by most men looked upon as involving an entire organ or tissue. Not knowing the microscopic structure they were not aware of the fact that all tissues are composed of varying kinds of cells. It was Virchow, the celebrated pathologist, who was the first to point out that all abnormal changes *en masse* are originally variations in the activities of the individual cells. The highly complicated structure of the human body had its beginnings as a single cell. This one cell contained within it all the potentialities of all the specialized cellular activities. As this cell undergoes division by the very complicated process of karyokinesis into hundreds of other cells, the first formation of importance is the division into three layers, the ectoderm, the mesoderm and the entoderm. It is from these three that the different structures of the body develop. It is from the ectoderm that the epithelial coverings of the body are formed, from the mesoderm come the bones and the connective tissues, while from the entoderm come the cells that line the great cavities of the body and also many of the internal organs. Inasmuch as the teeth develop from the outer and middle layers, certain malformations may be traced back to disturbances occurring in the very earliest stages.

As the abnormal changes of the body must be traced back to single cells it can be shown that a study of lower forms of life will frequently assist in an understanding of the more complicated phases of human pathology. If some single-celled form of organism, such as an ameba be studied, there will be noted certain activities that together constitute the manifestations of life, and which are considered to be the criteria of living matter. One of the most important of these is irritability which is that property of living substance by means of which there is a response to stimulation. Even in this earliest form of life it will be found that the cytoplasm, or cell substance, has the power of indicating likes and dislikes. It shows that there is some controlling force by means of which the cell is enabled to act in an independent manner.

The external irritants, or stimuli, may be of various kinds. It may be a change in temperature. Most of the pathogenic, or disease producing organisms, with which we deal grow best at about 37½°C., the normal heat of the body. There are, however, certain thermophilic bacteria which grow at 80°C. while the spores of some can resist such variations as 100°C. above to 225°C. below O°C. In this study such great variations do not concern us as the range of temperature that is compatible with human life is but a few degrees.

In addition to the above form of irritation it will be found that living protoplasm will respond to mechanical stimuli. This is seen when sterile fragments of foreign substance are placed within the body. There will be a response by the tissues which will attempt to dispose of the material. Light and electricity are also powerful stimuli and can produce most serious derangements.

In comparing these manifestations of the unicellular organism with those of the human being, it will be found that there are direct analogues in the higher form of life. In discussing the subject of inflammation, some of its phenomena are those that are of daily occurrence in the lower form. At first glance it might seem that there was very little in common between an ulcer in the body of a human being and an ameba gathering food for itself, yet identical activities take place. One of the necessities of living matter is an ability to obtain food of the proper kind. An ameba has the power, when it finds suitable food, of sending out prolongations of its cytoplasm which eventually will surround the substance desired. This will become enclosed in

the body of the cell, undergo digestion and be absorbed, any waste material being cast out. Under the microscope this most interesting process can clearly be seen by placing in a drop of water containing ameba some minute forms of life which will attract the unicellular protozoa. The motion of the ameba is very slow but by a flowing of protoplasm progress across the microscopic field takes place. When it comes into contact with the bacteria, a slight protrusion of the surface occurs on both sides of the morsel and extends slowly until there is a distinct depression in which the food substance lies. These prolongations or pseudopods continue to increase until they come into contact on the further side of the engulfed body. Then by some vital process these extensions fuse, the ameba returns, more or less, to its spherical shape and proceeds to digest the material. An identical process is that which is found in the human body at most times, but particularly in inflammatory areas. In the blood there are found two main types of cells, the red whose function is to carry hemoglobin, and the white. Most of these latter possess the ability, to a great degree, of the ameba to gather up foreign particles. This activity is known as phagocytosis and its chief exponent is the polymorphonuclear leucocyte. When bacteria gain entrance to the tissues, the phagocytes are the main line of defense, as on account of the attractive force or chemotaxis, the leucocytes will pass out into the invaded area. When there they will attempt to take up the alien newcomers and absorb them. Unfortunately the leucocyte may take up more than it can handle, it cannot digest all that has been taken up. The result is that these cells with the contained bacteria may find their way back into the circulation and in that way give rise to secondary troubles in distant parts.

The phagocyte, however, plays a more important role in the protective mechanism of the body. According to Metchnikoff it is the phagocyte that is particularly concerned in immunity as when these cells break down there are liberated very active enzymes or ferments which act upon the invading organisms as well as degenerated tissue.

When the various steps of inflammation are discussed it will be seen what a leading part is played by these same leucocytes.

In the study of disease in any part of the body a fundamental requirement is a thoro knowledge of those processes which together constitute inflammation. There are so very few conditions in which it does not play a large part that they can almost be omitted. No matter what the original lesion may have been, inflammation sooner or later comes to the front.

Before, however, taking up the relation that exists between inflammation and injury to the tissues it will be necessary to consider the changes that occur in the process of inflammation. We are accustomed to consider the so-called cardinal symptoms, pain, swelling, redness, heat and altered function as essential for a diagnosis. It must be remembered tho that all these changes may be present, yet in so slight a degree as not to be recognized by the naked eye, but, nevertheless, giving rise to appreciable disturbances.

In order to have a working basis, the causes of inflammation may be divided into the infectious and the non-infectious. The first type resulting from the presence of a living organism, and showing a more or less marked tendency to extend not only in the immediate neighborhood but to more distant parts. The second type, not being due to a living organism, tends to remain local and self-limited.

When the inflammation is due to the presence of bacteria, there is added the

complication of a living structure, foreign to the host. It is not so much that the living invader does harm by its mechanical presence, but more as a result of its metabolic processes and its disintegration products, both of which are made up of many forms of chemical substances. These are the causes of the cellular reactions, and according to the degree of their toxicity, the inflammation will be more or less severe. The compounds set free by bacterial activity may exert either a general or a local effect, or the organisms themselves may be carried by means of the blood circulation into more distant parts of the body and thus cause secondary disturbances.

Of the non-infectious inflammations there are those due to the action of chemical substances other than those derived from living organisms, and those due to more mechanical causes. Altho primarily no bacteria may have been present, as a rule, they sooner or later make their appearance, and in this way complicate the picture. It is very seldom that a pure non-infectious inflammation remains as such for any length of time. Consequently it is justifiable to conclude that by the time an inflammation comes to the attention of the practitioner there are bacteria present which must be destroyed or their products neutralized.

Granting the presence of an irritant, particularly one of microbic origin, what are the changes that take place in the tissues? Altho almost without exception the vast majority of the tissues are vascular, yet there are certain structures that do not have a blood supply. In order to trace the manifestations of inflammation from the simplest to the more complex it will be advisable to start with a non-vascular area. In the higher forms of life such a structure is the cornea. This is formed of fibrils arranged in layers lying parallel to the surface. The different layers are at an angle to those above and below and no blood vessels are present. There are, however, numerous channels thru which the lymph flows readily. Between the layers are found spaces in which the flattened connective tissue cells of the structure lie. If a strong solution of zinc chloride is applied and washed away before there has been any destruction of the corneal layers the only indication of any injury or reaction will be the breaking down of the corneal cells immediately beneath the injured area. No leucocytes will be present. Repair therefore can take place without the presence of any of those particular cells, nor will there be any of the ordinary phenomena of inflammation.

When, however, the corneal tissue is broken, there will develop within a few hours a whitish opacity which will be found to consist of a massing of small round cells that on careful examination will prove to be leucocytes. The peripheral blood vessels will not show any dilation. The leucocytes have entered the wound from the cornea itself, from the conjunctiva and the lachrimal fluid bathing it. In this experiment the inflammatory process is represented by the destruction of tissue and immigration of leucocytes, followed by repair. Neither the vascular nor the nervous systems play any part.

If instead of the injury being a sterile one, pyogenic cocci are used, it will be found that the organisms multiply and cause a degeneration of the fixed tissue cells. Within eighteen hours the leucocytes make their appearance. Within twenty-four hours there is a dense packing of leucocytes around the central degenerated area and a great number of these cells may be seen converging along the lymph spaces from the periphery of the cornea. The veins at the periphery become more prominent, the area has a congested appearance, the smaller as well as the larger vessels are dilated and

the leucocytes are passing cut from the contained blood into the surrounding lymph spaces. From the blood vessels at the periphery fine new vessels with very delicate walls pass toward the injured region and active repair goes on.

As the changes in the avascular structures are studied, it is seen how they lead up to the disturbances occurring in tissues that have a blood supply. This supply consists of a greater or less number of arteries, veins and capillaries. If these different vessels be examined in a living state under the microscope, it will be found that they present very definite characteristics.

In the arteries the stream is not constant; it is regularly intermittent on account of the rhythmic contractions of the heart. It is more rapid than in the veins and at the height of the systole the red cells cannot be distinguished, but at the end of the heart's action the current slows sufficiently for them to be seen. The corpuscles and the plasma are thoroly mixed and the cells will be found in the entire lumen, except that at the end of the pulse wave they momentarily withdraw from the wall of the blood vessel.

In the veins the stream is constant and regular in speed. Instead, however, of the cells and plasma being intimately mixed there are two zones present. Of these, the axial, or central is composed mainly of blood cells. The peripheral zone is made up ot plasma in which a few leucocytes, but no red cells, may occasionally be found.

In the capillaries the current is neither constant, nor regularly intermittent. It is constant as long as the flow continues.

As a result of the introduction of an irritant, certain very definite phenomena result. If the cause has been applied suddenly there is usually a momentary contraction of the blood vessel. But by the time the inflammation has become clinically apparent the chief change noted is a marked dilatation and relaxation of the vessels. This is first noted in the arterioles, then the veins and capillaries. There is probably a very temporary increase in the rapidity of the current but as the dilatation continues there follows a slowing of the blood stream. Instead of the cells in the arteries being unrecognizable, they now become distinctly visible. Marked changes now take place, particularly in the venous circulation. The plasmatic zone, which normally contains but a few leucocytes, shows an increase in number until it is entirely filled by them. Following this there takes place an escape of cells and fluids from the vessels.

At first the leucocytes adhere but slightly to the walls of the blood vessels, assuming a pear shape, the enlarged ends extending in the direction of the current. In the course of five or six hours all the small veins of the involved area may show a mass of leucocytes along their walls. These in time become closely attached, and finally many of them may pass thru the vessel walls into the surrounding tissues. This process is known as emigration, but the mechanism of its occurrence is not thoroly understood. The phenomena can hardly be due to nervous influences as the changes take place too slowly. It is also impossible to bring about an inflammatory reaction by stimulating either the vaso-constrictors, or the vaso-dilators. When the latter is done there is an escape of plasma but not of the cells. Probably the chief reason is that the ameboid motion of the leucocytes is very much stimulated. It may be the result of positive chemotaxis, the attraction that certain substances exert upon motile cells. Dead tissues and the products of bacterial growth are positively chemotactic, and their influence may be exerted upon the leucocytes while these cells are still within the blood vessels.

In addition to the emigration of the

leucocytes, diapedesis, or the escape of red cells may take place.

At the same time that the cells escape there is an exudation or outflow of lymph thru the vessel walls. This increased amount of lymph renders the tissues swollen and edematous, and gives room for free ameboid movement of the leucocytes, many of which are actively phagocytic.

As a result of the disturbances of the circulation there are those changes in the inflamed part as a whole that have already been referred to as the cardinal symptoms. The redness is due to the hyperemia, the excess of blood in the part. This increase is important as there are brought to the involved area more leucocytes, diluents and antibodies, and in this way facilitates the removal of harmful substances, and in some cases possibly affords increased nutrition to the cells.

The increase in temperature is probably the result of two causes, one that more blood is brought to the part, the other that as the blood moves more slowly, the heat accumulates. It is also a recognized fact that oxidation and other chemical activities occur more rapidly in the presence of heat.

The pain is due not only to the pressure exerted upon the terminal nerve filaments, but also to the action upon them of toxins, acids, enzymes, etc.

The swelling is the result of the increased amount of blood and the presence of the exudate.

This exudate may be one of several varieties or there may be a combination of different forms. The simplest type is the serous which is composed of fluid that has escaped from the vessels and which contains but few cells. This fluid differs from the non-inflammatory transudate in that it contains a greater amount of albumen, therefore being of a higher specific gravity and tending to coagulate spontaneously.

Another form is that in which fibrin is present, this substance probably helping to restrict the dissemination of the infecting agents. Its formation need not be gone into as it is not a matter of practical importance.

If there is a preponderance of escaped leucocytes the exudate is referred to as pus, and as such may be found either infiltrating the tissues or remaining circumscribed and forming an abscess. This matter of local or general involvement is due largely to the peculiarities of the different bacteria. When infection by streptococci takes place, the common tendency is for the organisms to infiltrate widely the involved structures, very little attempt at localization being found. On the other hand when staphylococci infect, the usual result is a more or less distinctly localized area known as an abscess.

As abscesses are of particular importance as a consequence of infection about the teeth, a description of the various steps occurring in their formation may be given. After the pus producing bacteria gain entrance they undergo multiplication without at first causing any reaction. In a very short time, however, the invaded area becomes congested, the leucocytes approach the walls of the blood vessels, and degenerative changes in the neighboring tissue cells appear. The multiplication of the bacteria continues, the polymorphonuclear leucocytes escape from the vessels, and the small round (mononuclear) cells collect. The polynuclear and other cells, endothelial in origin, take up large numbers of bacteria. More leucocytes appear until the tissue become densely filled by them. This is accompanied by a yet greater multiplication of the organisms which extend along the lymph streams into the region outside of the developing abscess. There is now a breaking down of many of the escaped leucocytes with the setting free of various enzymes

which assist in bringing about a coincident destruction of the involved tissues.

Fortunately inflammation does not necessarily or even usually result in abscess formation. Its termination depends upon the degree of the inflammatory changes, and the amount of damage that has occurred. The simplest outcome is recovery by resolution. This occurs only when the reaction has been slight and very little damage done. The exudate is taken up by the lymphocytes and returned to the circulation. Any degenerated cells will be removed by the phagocytes and the tissue will resume its normal condition.

If, however, the inflammation has gone on to suppuration, pus formation, the termination is very different, it being a much more serious condition. As pus is formed, it is either confined as an abscess, or else it tends to infiltrate the tissues. In either case the tissues attempt to get rid of the irritating substance by having it follow along the least resisting path and having it escape from the body. This process of extension is known as burrowing. It results from the increased pressure due to the presence of the pus and to the digestive powers of the enzymes present. In some cases the pus may escape quickly to the surface of the body and be cast out. It may, however, have to burrow a long distance before it can escape. During the process of formation and extension there is always the danger that the infecting organisms may gain entrance into the circulation and give rise to metastatic or secondary abscesses elsewhere in the body. It is this possibility that makes the question of focal infections of such great importance. If one could be sure that an apical abscess would remain as such and not give rise to trouble in other parts of the body, such a lesion might be considered as negligible. The unknown possibilities of such an area are what cause the uncertainty. In general surgical practice it is considered the proper thing to open up an abscess, clean out the cavity and allow it to heal as an open wound. Unfortunately the proposition is a very different one when the teeth have to be taken into consideration. Removing the tooth is a comparatively simple affair. By so doing the cavity is exposed and it readily responds to treatment but the patient has lost an important structure. The question, of course, becomes more serious when several teeth are involved.

In other parts of the body the usual end of an inflammatory reaction in which more or less destruction of tissue has occurred is by organization or repair. In this process there is formed what is called granulation tissue which acts as a strong barrier to absorption and reinfection. This tissue, so-called, is one that is seldom recognized as being of such great value in maintaining the integrity of the body. The surgeon speaks of healing by first and by second intention and will try always to have his wounds recover by the former. Yet the process in the two is the same except in the matter of quantity formation. Healing by first intention is that which occurs when the area is not infected. When, however, bacteria are present the injury is repaired by second intention in which there is the formation of a large amount of granulation tissue. This tissue is constantly bathed in secretions which tend to wash away bacteria, broken down cells and other debris. The amount of blood present is greatly in excess of that which is required by adult tissue, but at this stage of cellular activity much nutriment is needed. Ordinarily such an area is very resistant to infection, that is to the passage of bacteria thru the newly forming substance into the body. As a rule the granulations tend to fill up the greater part of the destroyed tissue by growing up from the bottom, while from the edges there

will be an outgrowth of epithelium, provided there is such immediately adjacent. When the defect is filled and the surface covered by epithelium, the healing process is complete, the physiological balance has been re-established and the activities cease.

The amount of new connective tissue found will depend directly upon the specialization of the destroyed cells and their ability to regenerate. It can be considered axiomatic that the more highly specialized the tissue the less able is it to be restored to the normal. When a nerve is severed there is probably little if any regeneration unless the ends are very closely approximated and held in position. On the other hand there seems to be almost no limit to the ability of surface epithelium to repair injury. Large areas of skin may be denuded yet the epithelium will grow across the exposed portion and restore it to normal.

There is, however, one particular type that is most active in repairing injury, the connective tissue cells are the living exponents of the saying that nature abhors a vacuum. Connective tissue cells are found everywhere in the body, altho in the central nervous system the glia cells, which form the supporting framework, do not have the same embryonal origin as those in the other parts of the body. Whenever a structure is damaged there is a varying degree of inflammation occurring, and, as a result of the protective reaction, the different cells in that area will attempt to regenerate according to their ability. Few of them will be entirely successful. The connective tissue then comes to the rescue, replaces the destroyed cells and the part recovers. It, however, probably will not be as efficient as formerly, but in many instances, particularly when secreting organs are involved, the remaining cells will hypertrophy. In this condition they will increase in size and possibly in number. By this means one of paired organs may be removed or destroyed, yet the individual be able to carry on a normal existence. To return to the question of granulation tissue, in it there is the formation of loops of new capillaries derived from the endothelial lining of pre-existing blood vessels. The endothelial cell becomes larger, the nucleus divides in two and two cells are formed. These cells continue to divide until a bud-like process extending into the surrounding tissue is formed. Adjoining sprouts unite and, altho at first solid, finally become hollowed out and the circulation becomes reestablished.

At the same time that this is taking place there is a multiplication of the fixed connective tissue cells, which surround and act as a supporting framework to the loops of the newly forming capillaries. In the proliferation of the connective tissue there is first found a small round cell with a round or oval nucleus. As the tissue becomes older the cells tend to elongate and become spindle shaped. At first they are very close together, but gradually separate, and the homogeneous intercellular substance becomes fibrillar and supports the cells. In the newly-formed tissue there is in the beginning an over-production of cells and blood vessels, but eventually it becomes less vascular and cellular. This is brought about mainly by the contraction of the new connective tissue.

Instead of either resolution or organization taking place, accompanied by the absorption of the abnormal products, the process of encapsulation may occur. This is what may happen when the irritating material cannot escape from the body. The surrounding tissue cells undergo multiplication and the broken down area is isolated by the formation of a connective tissue capsule about it. During the active stage of granulation tissue formation there is a direct and

rich communication thru the newly formed capillaries with the general circulation. This, nevertheless, does not mean that there is any great danger of infection. The tendency is all toward protection, and encapsulation is therefore a very efficient means employed by nature. As already stated, the capsule is made up of layers of dense connective tissue that offer great resistance to the passage of both bacteria and their products. It must be remembered, however, that the infectiousness of the lesion has not been destroyed. If for some reason the surrounding capsule is weakened or destroyed, the contained material may escape into adjacent tissues and set up a new inflammation; or it may gain entrance into the blood and be carried thruout the body. In tuberculosis this phenomenon is not uncommon but it may take place in any localized lesion.

Inasmuch as inflammation is such a common affair one can appreciate its necessity from the view point of its being a protective reaction. As stated in the beginning, it might be defined as a protective reaction of irritated and damaged tissues which still retain their vitality. For that reason care must be taken not to interfere too much with an inflammatory change. The first thing to do, of course, in any inflammation, is to remove as thoroly as possible the underlying cause. If the irritating material were purely mechanical in its action its removal would, as a rule, cause the abnormal condition to subside and the tissues to return to their normal state. When bacteria are present, the question is by no means a simple one, because as long as many micro-organisms remain they will continue to multiply. In so doing the production of metabolic excretions will continue and the inflammation persist. The activities of the practitioner must be directed largely toward getting rid of these invaders or at least to so interfere with their course that any products will be neutralized and the patient be undisturbed.

The practitioner will find in the examining and treating of his patient that there are certain factors that will influence his course of action. The degree of involvement will depend upon the virulence of the infecting organism, upon the resisting power of the individual attacked, and upon the tissue involved. According to the intensity of the virulence and the lack of the susceptibility it will be found that apparently similar infections will react differently upon different people. In considering an infection the general tendency is to forget that the location of the point of entrance has a great deal to do with the development of a disease. The usual idea is that when bacteria get into the body it makes very little difference as to the tissues primarily involved. A very good example of tissue susceptibilities is shown in tuberculosis. In many instances, particularly in children, the tubercle bacilli gain entrance by way of the intestinal canal. They pass thru its walls without doing any local damage, enter the lymphatic circulation, and are carried to the nearest lymph node where a tuberculous process is set up. This may remain limited for years. Again, it may be that in some way the tubercle bacilli get into the skin. Instead of being carried away by the blood or the lymph they remain and develop a local lesion. This may slowly increase in size till large areas are affected yet it does not spread elsewhere. On the other hand it is very evident that when these bacilli get into the lungs, the picture is a very different one. Here, in spite of a good blood supply, the bacteria will multiply, the tissues will break down, be cast out, and eventually large cavities may develop.

It is therefore evident, as above stated, that not only are individuals different in their susceptibility but that there are

even variations in the tissues in regard to their response to infection. In this connection it is interesting to note the marked variations of tissues in regard to malignant tumor formation. One of the striking points in a study of these growths is the very evident immunity that is possessed by certain organs. It is quite unusual to have a primary cancer of the liver, yet after a cancer has once become established elsewhere in the body, the liver is about the first structure to show secondary lesions. On the other hand, the spleen is very rarely the seat of malignant neoplasms. Another interesting fact is that cancer of the thyroid gland, an epithelial structure, very commonly gives metastases in the bony skeleton, which belongs to the connective tissue group. There are, indeed, many factors unknown to us which influence the manifestations of disease. One of the more important lines of work to be undertaken will be an attempt to determine by laboratory methods the virulence of a given organism and the susceptibility of the patient, and to correlate the findings.

It will also be noted that the outcome of the inflammation will depend upon the location of the lesion, whether it is superficial or deep, whether in soft tistues or in bone. All of these factors have to be taken into consideration.

As active practitioners the point of greatest interest is the application of the inflammatory changes to these structures which daily come under your care. In the light of what has been said already in regard to these changes, it will not be amiss to review the histology of the tooth pulp. By so doing it can be shown how the symptoms following an infection of this structure depend upon the inflammatory reactions as modified by environment. The pulp consists of an embryonal type of connective tissue, rich in branching stellate cells and poor in fibers. It contains no elastic tissue, and the delicate collagenous fibers, instead of forming bundles are arranged in an interlacing net work, the fine fibrils of which are in intimate relation with the connective tissue cells.

The stellate connective tissue cells are scattered thruout the entire pulp but at the periphery of the cavity are closely crowded and much enlarged. These peripheral cells form the layer of odontoblasts which are in contact with the dentin. The odontoblasts are cylindrical, branched connective tissue cells whose long axis is perpendicular to the surface of the adjacent dentin. From the apex there extends into the dentinal canals a delicate process which frequently passes all the way thru the dentin. Lateral processes from the cell bodies of the odontoblasts interlace with each other and finally unite the cells into a membranous layer. Other processes are given off from the base of these cells and intermingle with the fibers of the pulp, so that if this tissue is forcibly separated from the dentin, the odontoblasts remain adherent to the connective tissue of the pulp.

The dental pulp is richly supplied with blood vessels derived from a nutrient artery which enters thru the root-canal, its several branches forming a net-work of arterial and capillary vessels in the center of the pulp cavity, and a peripheral close-meshed capillary network which is in close relation with the layer of odontoblasts.

A rich nerve supply is derived from the fine branches which also enter by the root-canal. Most of the nerve fibers lose their myelin sheath soon after they enter the pulp. They form a primary plexus in the connective tissue from which they pass to the pheriphery and form a marginal plexus beneath the odontoblasts. From here delicate terminal sensory filaments ramify over the odontoblasts and pass in great abundance into the dentinal tubules, usually

two to each tubule. This peripheral distribution explains the extreme sensitiveness of the dentin.

Such a tissue as the above when undergoing the changes incident to inflammation will naturally give rise to reactions and symptoms more or less modified by the peculiarities of its structure and its surroundings. It is well known that the severity of the pain accompanying inflammation is greater in those areas in which the tissues do not have room to expand. A felon is particularly painful because it is a sub-periosteal affair and there is no room for the usual swelling. A similar condition, but in a greater degree, occurs when the pulp is inflamed.

The inflammatory changes explain why there should be pain resulting from the involvement of the pulp. As the result of the presence of an infecting organism, the blood vessels, which have very thin walls, become engorged. There is also an escape of cells and fluids from the vessels, all of which causes an increased pressure upon the odontoblasts and the net-work of nerves so intimately connected with them. If an opening is made into the pulp cavity, the pressure is relieved, products of the inflammation escape and the pain practically disappears. The sequence of events is that which follows the opening of an abscess.

The after treatment is, however, very different. The abscess cavity is kept open and allowed to drain. During this time there has been a constant formation of granulation tissue in the bottom of the lesion. This progresses until the cavity has been completely filled. If the destruction has been in an epithelial area the neighboring epithelium, as before mentioned, will cover the lesion and the process subside.

In the case of the teeth the difficulty lies in the retention of the diseased organ and at the same time rendering the apical abscess and the remnants of the pulp innocuous. It is here where the greatest diversity of opinions arises. Will the infected areas become sterile spontaneously or must some form of treatment be discovered that will insure sterility? There are many advocates of the utility of medicaments variously applied. There are others who will strongly maintain the futility of such treatment, and will insist that the teeth must be extracted. The latter class claims that as long as such an infected area remains there is always a great possibility of a systemic infection occurring. There is no doubt that focal infections are a very common source of generalized disturbances, but it remains to be proven that all, or even the majority of such foci ever cause trouble.

THE TOOTH BRUSH AND METHODS OF CLEANING THE TEETH.

A Report to the National Dental Association by Jules J. Sarrazin, President, the American Academy of Periodontology, New Orleans, La., Reporter.

(Read before a General Session of the National Dental Association at Its Twenty-third Annual Meeting, New Orleans, La., October 20-24, 1919.)

A COMMITTEE has undertaken the preparation of this report in response to the Academy's request that it present a description of a tooth brush of approved design and a method of brushing the teeth which will prove of service to the general practitioner of dentistry in instructing patients in the personal care of the teeth.

The committee has secured from the members of its Council opinions resulting from their clinical experiences, and has based its report upon such collective views, after also embodying those resulting from a general meeting of the membership of The American Academy of Periodontology.

The effort has been to attempt the standardization of the personal care of the mouth by recommending a brush of an accepted design, which may be generally adopted, and an efficient method of brushing the gums and teeth which may be universally applied. Both brush and method have been demonstrated by members of the Academy to be efficacious and non-injurious.

In recommending one form of tooth brush and one method of brushing the teeth, the Academy does not wish it understood that it discredits *all* brushes of dissimilar design or *all* methods of brushing the teeth which do not conform to the method outlined, but, that it endorses a brush and a method offering the greatest advantages.

The opinion has been unanimously expressed that a brush larger than one and one-half inches in length in the brush head, and what is known as the crosswise or mesio-distal stroke method, are both injurious and inefficient.

With this statement of its objects the report is presented to the National Dental Association with a request for its adoption.

The Tooth Brush.

For Adults:

Brush Head.

Total length of brush head, 1¼″.

Number of bristle tufts in row at neck, at most, 3.

Number of bristle tufts in row at extremity, 2.

Space between rows, 3/32″ to ⅛″.

Length of bristle tufts at neck, ⅜″ to ½″.

Length of bristle tufts at end, ¼″ to ⅜″.

Bristles in tufts should be rounded.

The brushing surface should present a slanting straight line from the neck to the end of the brush head, and it should be convex from side to side.

Bristles should be stiff.

Handle.

Should be fairly rigid.

Should be 6 inches (entire length of handle and brush head.)

The neck should be long and slender.

For Children over three years of age:

Brush Head.

Total Length, 1″.

Number of bristle tufts at neck, 2.

Number of bristle tufts at extremity, 1.

Otherwise, with the exception of a general decrease in size, the child's brush should resemble the adult brush.

For Children under three years of age:
Brush Head.

Total length, 7/8″.

Single bristle tufts set lengthwise of brush head, 5.

Length of bristle tufts at neck, 5/16″.

Length of bristle tufts at extremity, 3/16″.

Otherwise, with the exception of a general decrease in size, it should resemble the first child's brush described.

METHODS OF BRUSHING THE TEETH.

The American Academy of Periodontology recognizes four effective methods of brushing the teeth, known as:

1. The rolling stroke method.
2. The up and down stroke method.
3. The circular stroke method.
4. The vibratory method.

METHOD (1). The *rolling stroke method,* which takes *precedence over other methods,* consists of placing the brush in the mouth, bristle ends *up,* for the upper teeth, and *down* for the lower teeth, and rolling the brush toward the gums, sweeping the bristle over the gums and teeth to their occlusal surfaces.

This motion brushes the buccal and labial surfaces of all the teeth above and below, and the lingual surfaces of the molars and bicuspids in both arches.

The lingual surfaces of the incisors and cuspids are brushed by placing the brush parallel to the tongue, with the bristle ends on the roof of the mouth for the upper teeth and on the floor of the mouth for the lower teeth, drawing the brush forward over the gums and teeth to their incisal edges.

The upper and lower jaws are brushed separately.

The occlusal surfaces are brushed with a forward and backward, and side to side stroke of the brush.

METHOD (2). The *up and down stroke method* is presented as a practicable compromise between the rolling and the circular stroke methods, as one whose technic can be readily mastered by many whose personal mouth care might otherwise be less efficient. It consists of placing the brush with the bristle ends toward the gums and while in that position of carrying the brush down over the gums and teeth to their occlusal surfaces, and up again for the upper teeth, and the reverse for the lower teeth.

The buccal and labial surfaces of the upper and lower jaws may be brushed separately, or the teeth may be placed edge to edge and both upper and lower jaws brushed at the same time.

The lingual surfaces are brushed in the same way.

The occlusal surfaces are brushed with an in and out, and side to side stroke of the brush.

METHOD (3). The *circular stroke method* consists of placing the brush with the bristle ends toward the gums, and with a circular motion brushing the gums and teeth. The buccal and labial surfaces of each jaw may be brushed separately, or the teeth may be placed

edge to edge, and both upper and lower jaws brushed at the same time.

The lingual and occlusal surfaces are brushed with the same circular stroke.

METHOD (4). The *"vibratory method"* of brushing the teeth consists of placing the brush horizontal to the long axes of the teeth with the bristle ends resting on the tooth surfaces. When pressure is applied to the brush and a very short mesio-distal stroke executed, the bristle ends enter the embrasures between the teeth covered by the brush. The stroke, if it may be called a stroke, is so short that it does not carry the bristles beyond the teeth upon which they rested before the stroke was begun.

In this manner all the surfaces of the teeth are brushed.

METHOD 1 is a simple and effective method favored by the great majority of the members of the American Academy of Periodontology.

The gums and teeth should be brushed before breakfast and after meals. The most important time to brush is at night.

Those portions of the individual mouth which demand the most care should be brushed first. In general, the following rotation may be followed:

Lower jaw.

Lingual surfaces of the gums and teeth.

Buccal surfaces of the gums and teeth.

Labial surfaces of the gums and teeth.

Upper jaw.

Buccal surfaces of the gums and teeth.

Labial surfaces of the gums and teeth.

Lingual surfaces of the gums and teeth.

Occlusal surfaces of both the upper and lower teeth.

The brush should be rinsed frequently in clear water during the brushing.

The tongue should be brushed in the morning.

The brush should be thoroly rinsed after its use, and be placed where it will dry.

FLOSS.

Waxed ribbon floss should be used after brushing the teeth and gums. The majority of the members of the Academy recommend its use at night. It should be carefully rubbed over the proximal surfaces of the teeth and should not be allowed to injure the gums. The distance between the finger ends, when the floss is used, should be at all times reduced to the minimum, approximately one-half to three quarters of an inch. The floss should be stretched taut over the ends of the fingers, or finger and thumb, not wound around them.

Special care must be given to the proximal surfaces of teeth bounding spaces where one or more teeth are missing. These surfaces may be brushed with a single row bristle brush, where the space permits, or they may be cleansed with ribbon floss.

Ribbon floss threaded on a bodkin and passed between the dummy teeth and the soft tissues should be used to cleanse the under surfaces of fixed bridges and the abutment teeth.

INSTRUCTIONS INTENDED FOR THE LAYMAN.

BY THE CLOCK.

Brush your teeth for 5 minutes, 3 or four times each day.

1. Before breakfast.
2. After breakfast.
3. After luncheon.
4. Before going to bed.

Use a *small* brush.

Brush the *gums* and teeth.

Be *sure* to *reach both sides* of the *rearmost molars.*

TO BRUSH THE TEETH.

Place the tooth brush, ends of the bristles *up,* for the upper teeth, between the cheek and the gums; turn the brush

toward the gums, sweeping the bristles *down* over the gums and teeth to the cutting edges of the teeth. Reverse for the lower teeth and brush *up*.

This motion brushes the cheek side of all teeth and the tongue side of the back teeth.

Brush the inside of the front teeth by putting the brush, bristles up, on the roof of the mouth for the upper teeth, and on the floor of the mouth for the lower teeth, pulling the brush outward over the gums and teeth.

Brush the grinding surfaces of the teeth with an in and out and side to side stroke of the brush.

Rinse the brush in clear water frequently while brushing the teeth and hang it up to dry when not in use.

A tooth powder, paste or a solution of common table salt may be used as a dentrifice once each day. Use clear water at other times.

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FUNDAMENTALS OF PERIODONTOLOGY.

By John Oppie McCall, D. D. S., Buffalo, N. Y.

(Read before the District of Columbia Dental Society, May 20, 1919.)

STUDY of the diseases of the pericementum and associated structures requires first, consideration of these structures in a state of health and the factors responsible for that state of health. We must then have some understanding of the nature of the diseases which may attack these tissues, with an orderly classification of these disorders: this involves the employment of an adequate and scientific nomenclature. Etiology should next receive its share of attention, and we are then ready for diagnosis, prognosis and treatment.

The mouth in a state of health has been considered in a clear and detailed manner in a paper presented some time ago by Dr. Paul R. Stillman, and it will be necessary here only to call attention to the salient aspects of the healthy mouth, and to mention the chief barriers against gingival disease which are the birthright of each one of us. The pink lustrous appearance which denotes the healthy gum tissue is most frequently seen in the mouth of a child, where too we see the septal spaces filled to the very contact points of the teeth by the crests of the gum septa. The deciduous dentition too, more frequently than the permanent, exhibits a practically normal occlusion, but may not always show that cleanliness which means not only freedom from irritants and bacterial masses, but implies stimulation of the circulation. We are too apt to ascribe this condition of health to Nature, without thought as to the foundations upon which it is built, and to blame Nature in a equally vague way when the normal begins to give way to the pathological. We must first of all realize that a state of health is due to certain definite factors, and that a state of disease is due to a disturbance of these factors as well as to the introduction of disease-producing agencies.

The health of the peridontium depends first of all on normal blood supply, both in the amount brought to and carried away from the part, in the proportions of nutrient and protective elements in the blood itself; and also on a normal lymphatic circulation. The tissue cells, too, must be in a condition which will lead them to assimilate the nourishment brought to them; for while we can lead a cell to the blood stream, we cannot make it drink. This condition depends partly on tonicity, partly on nerve reflexes. The next in our items of health foundations is normal occlusion, normal not only as the orthodontist sees it on his articulated models but coordinated in its antagonization no matter what the manner of closing or moving the jaws in mastication. Then comes the clean smooth tooth surface, which can only be maintained by the friction of food well chewed, or by the

vigorously wielded tooth brush. These are your fundamentals of the healthy mouth,—normal blood supply, normal occlusion and cleanliness. Pathogenic agencies can gain no foothold in the mouth where these conditions prevail.

The periodontium is made up of pericementum, alveolus, alveolar gingiva and marginal gingiva, which last includes the interproximal gum margin. Diseases which attack these tissues are grouped under the title "Periodontoclasia," this term meaning a destruction of the tissues immediately surrounding the teeth. The subtypes of Periodontoclasia or Dental Periclasia are classified largely according to the tissue which is the center of the attack. Analysis of these disease conditions reveals the fact that we are dealing with several fairly distinct types, and that each case is usually a composite of two or more of these types. Thus we have Recession; Gingivitis; Pericementoclasia, to which type the name Periodontoclasia in a limited sense may be applied; Alveoloclasia, and Periodontal Abscess. Once these basic types are firmly fixed in one's mind, diagnosis becomes a matter of noting the tissues involved and the character of the disturbance.

Recession and Gingivitis are confined largely to the gum margin. Pericementoclasia is primarily a destruction of the pericementum, thus leading to the formation of the so-called pocket; it, of course, involves destruction of alveolar bone, but this is a secondary phenomenon in this type. The outstanding feature of Alveoloclasia is the absorption or destruction of alveolar bone without corresponding loss of the pericementum. In this type there is no pocket formation, yet the tooth exhibits mobility due to loss of bony support. Periodontal abscess needs no definition. It has two main sub-divisions,—apical originating from a pulp disturbance and with which we are not at present concerned; and parietal, not of pulpal origin, but located either in the pericementum at the side of the root or in the gingival tissue overlying a previously formed pocket. Having adopted these types, which admit of suitable subdivisions, we can say for instance that this tooth exhibits Gingivitis and Suppurative pericementoclasia, while that one shows Recession and Alveoloclasia. This avoids the necessity obtaining in past years of saying that both cases are pyorrhea alveolaris, and trying to figure out how two cases can show conditions so totally different if it is all one disease.

Just as we find variations in the types of periclasial disease, so we naturally find variations in the pathology and etiology of the cases coming under our observation. The pathology of a simple recession is vastly different from that of a suppurative pericementoclasia, just as the etiology of gingival abscess differs from that of alveoloclasia. The etiology of periclasia, like periclasia itself, is a composite consisting always of two or more factors, often widely different and varying in their relative importance from case to case, but always a combination of two or more forces working together for the destruction of periodontal tissues. Any one of these factors may be found alone, and will when so found, prove to be relatively harmless. Thus we find that calcareous deposits do not invariably produce periclasia. Bacteria are present in every mouth, and yet the most neglected mouths may show a healthy periodontium. Even traumatic occlusion may occasionally be seen in mouths which show no evidence of disease. But let us combine calculus, bacteria, traumatic occlusion and lowered tissue vitality, and we invariably produce a suppurative pericementoclasia.

These etiological factors may most

conveniently be arranged in six groups as follows:

1. Malocclusion.
2. Defective operative and prosthetic procedures.
3. Trauma.
4. Abnormality of tooth surface.
5. Abnormal systemic conditions.
6. Parasitic invasion.

The first four are called primary or inaugurating factors, while the fifth and sixth are secondary or non-inaugurating.

There is as previously mentioned a disposition on the part of periodontists to consider the purely local factors as the most important, or in other words the determining factors, while systemic conditions and bacteria are assigned a secondary role in bringing about the inception of periodontoclasia. In apparent opposition to this, Hartzell in a recent paper states emphatically that periodontal disease is purely a bacterial proposition; that to eliminate periclasia it is only necessary to eliminate bacteria. Careful examination of the two view points shows them to be far from irreconcilable, especially when we stop to analyze Dr. Hartzell's means of prevention. He recommends prophylactic treatment and the most vigorous home care as a means of reducing to the lowest attainable point the number of bacteria on the gingival third of the tooth surface. He admits that not all pathogenic bacteria can be scrubbed off in this way, but fails to call attention to the reason why the few remaining organisms are incapable of producing disease. This reason does not lie in their small number or lack of pathogenicity alone but in the resisting power built up in the gingival tissue by the massage of tooth brush and orange wood point. Dr. Hartzell also fails to take into account in this essay, the fact that we have types of periodontoclasia in which bacteria play no important etiological part, either primary or secondary, namely recession and alveoloclasia. Your essayist believes that it is more rational to give the etiological place of prominence to the factors which pave the way for the bacterial invasion and not to the bacteria themselves. In the final analysis nature must fight off micro-organisms by her own strength. We can never keep the mouth free from bacteria for any length of time, and when they have gained a foothold in the tissues we can never by methods of sterilization destroy quite all of them,—Nature must finish what we begin. Moreover we find that when we have perfectly removed the irritants which your essayist believes should be called primary factors, the protective forces of the blood will in most cases put the bacterial army to rout with little or no assistance in the way of artificial disinfecting agencies. So while it is absolutely accurate to say that bacteria are responsible for pus formation and tissue destruction, be sure to keep your perspective undistorted,—remember that your fundamentals in etiology are malocclusion, operative shortcomings, abnormal tooth surface and trauma, and that bacteria and physical ailments while important are usually secondary in their relationship to other factors.

The pathology of the various types of dental periclasia shows variations, as is to be expected in disease conditions exhibiting such striking diversities. We must bear in mind of course that these types are seldom found in a pure or uncombined state; it is much more common to find two or more types blended, and with lines of demarcation so indistinct as to furnish ample excuse for the jumble of opinions advanced in the past. In the same way we find a blending and intermingling of the pathology of the

various types. We will therefore consider the pure types, with the understanding that composite cases will show a corresponding composite pathology.

Recession is a condition characterized by a diminution of the tissue elements of the marginal gingiva together with the crest of the alveolus. It involves the severing thru atrophy of the gingival pericemental fibers. There is no inflammation in the usual sense of the word. There is little evidence of bacterial invasion.

Gingivitis presents the characteristics of the true inflammation,—redness, swelling, often with hypertrophy, presumably heat, and conscious or subconscious pain. There is usually additional evidence of parasitic invasion as indicated by the character of the secretion, odor, and possible pus formation. There is little, if any destruction of pericemental fibres, or alveolar bone unless the element of pressure enters into the etiology as in the impingement of crown bands or unconfined amalgam. Remember always we are speaking of pure types.

Pericementoclasia, because the pocket which is characteristic of this type provides so excellent a breeding place for bacteria and because of the character of the circulation and lowering of resistance, is practically always suppurative. If the amount of pus is so slight as to require microscopic demonstration, we may in a relative sense, call the type non-suppurative. The pathology of suppurative pericementoclasia is characterized by hyperemia of the blood vessels of the pericementum produced by the primary irritant. This progresses thru passive hyperemia, congestion and stasis. We now find the resistance and tone of the tissue lowered, making easy the invasion of the pericementum and alveolus by the ever-present micro-organisms; and we also have as a consequence of congestion and bacteriolysis, a breaking down of the capillaries, with a resulting hemorrhagic exudate under the free gum margin and the formation of serumal calculus on the tooth surface. Bacteriolysis continues with digestion and severing of the pericemental fibers. The deposited serumal calculus now adds its irritation to that of the inaugurating cause, with a corresponding aggravation of the previously mentioned phenomena. To the lowering of tone due to local causes we may now find added the depressing influence of some systemic condition tending to increase the rapidity of destruction or influencing the morphological or other aspects of the lesion. In this manner there is frequently established a vicious cycle, since the mouth lesion will in its turn inevitably influence to some extent the systemic disorder.

The pathology of alveoloclasia is characterized by an absorption of the bone of the alveolus due to excessive pressure transmitted thru the pericementum. There is usually little or no irritation of the marginal gingiva by deposits etc. As a consequence of this and a high cell vitality the resistance to parasitic invasion is high, hence there is little if any severing of pericemental fibres, and no pocket formation, altho there is usually some recession of the gum margin due probably as much to the absorption of the alveolus as to bacterial action.

Parietal periodontal abscess may be of the type occuring in a previously intact pericementum. It is then spoken of as a pericemental abscess, and is of hematogenous origin. A previous irritation, often traumatic, is the usual forerunner of the bacterial proliferation. This may result in a deposition of serumal calculus at some point along the side of the root without the cervical

fibres having been severed. This of course sets up the same cycle of pathological events found in pericementoclasia. The parietal gingival abscess is located in the gingival tissue overlying but not attached to the root surface, and is therefore always preceded by a pericementoclasia. Its pathology is therefore essentially that of the previously existing periclasia.

Diagnosis is the guide-post to treatment. Without intelligent differentiation of types of disease, and detection of the factors responsible for their occurrence we are reduced to the treatment of symptoms rather than treatment of the disease itself, a decidedly empirical proceeding and one that is likely to be unsatisfactory to all concerned. The first step in diagnosis is the examination of the case and the recording of conditions found and their etiology. The most careful examination may prove to be incomplete when its results are set down on paper, and while the taking of a complete case record consumes much valuable time, yet it is an invariable pre-requisite to successful treatment. The case record published by the American Academy of Periodontology is so arranged that it can be used for recording cavities, etc. as in the usual dental chart, and is provided with space for the recording of pathological conditions and their etiological factors. The conditions may be entered by key numbers in appropriate places on the diagram, or the tooth number may be entered in the tabulation record opposite the name of the condition found. The latter method has the advantage of showing at a glance all of the teeth in the mouth which show the same condition. The extent of recession may be shown by a line, the depth of pockets by a shaded area on the diagram, where also, the position of root fillings, rarefaction and similar conditions may be graphically recorded. The use of a case record of this kind is particularly urged for the general practitioner who sees practically all cases of periclasia in their incipiency. He looks over his patient's teeth, but usually overlooks the gums, until a decided pus formation or mobility draws his attention to a condition which has developed before his eyes, and over which in the majority of cases he could have exercised perfect control had he observed it while in the early stages of its evolution. A definite examination of the soft tissues should be made at regular intervals. Needless to say the first and slightest departure from a state of perfect health should be recorded, diagnosed and the causes responsible for its appearance remedied, for the periodontist in the future may not be as lenient as in the past, in fixing the responsibility for the inception of periodontal disease. The instruments used in examining the soft tissues are the usual mouth mirror, any rigid instrument as for instance foil carries for testing the mobility of the teeth, and a soft but firm blunt probe of silver or platinoid for determining the location and depth of pockets. Macroscopic pus formation will be determined by palpation with the finger or with a ball of cotton in the cotton carriers. Having analyzed the case to determine the type of periodontoclasia present, our examination is now directed toward the detection of the various etiological factors present. The essential feature of this examination lies in its completeness. To leave out one factor is to invite disaster. Bear in mind always the secondary part usually played by systemic conditions and parasitic invasion in etiology: diabetes and Vincent's infection offer instances in which these factors assume more nearly a primary role.

Prognosis requires the assembling and weighing of many factors. These

are, the vitality of the investing tissues and of the whole organism, the vitality of the cementum and underlying dentin, the proportion of intact pericementum and alveolus, the occlusion present or attainable, the amount of work the affected tooth must do, the possibility of properly curetting the denuded root surface, and the probable future care the mouth will receive. As to vitality, there are very few patients able to walk into a dental office, whose general physical condition will markedly retard a recovery from dental periclasia. More important is the morale of the tissues around the affected teeth; fortunately this is very largely under the control of the tooth brush. The tooth with a healthy vital pulp may safely be said to have a vital dentin and, except perhaps for a superficial denuded layer, a vital cementum. The tooth from which a healthy pulp has recently been removed and which has the inner ends of the dentinal tubuli properly sealed, may also be considered vital at least as relates to its cementum. The vitality of the cementum and dentin of other teeth must at the present time be left open to question. The proportion of intact pericementum and bone necessary for success is seldom less than one half; it varies with the amount of work the tooth must do and the leverage on the tooth. For instance a root having a nearly complete bony attachment on two surfaces is a better subject for treatment than a root having the same proportion of bony attachment distributed evenly around and confined to its apex. An innocuous and coordinated occlusion must be had or the most painstaking treatment will come to even less than naught. The tooth which has all or nearly all its quota of neighbors is a better subject for periodontic treatment than the one which must stand alone, unless prosthetic restorations can be made which will, not seemingly, but actually, relieve it of excessive labor. As to the future care of the mouth, prognosis depends not on divine power but on a scrupulous, intelligent and vigorous mouth toilet, supplemented at such intervals as may be found correct by thoro and discerning prophylactic treatment. Without the likelihood of securing such cooperation, the gathering clouds may not reveal their silver lining.

Treatment fundamentally consists in removing the cause of the disease and improving the tonicity of the part affected. The correction of most of the etiological factors mentioned, needs no detailed explanation, the great difficulty lying in detecting all the factors present and in securing an improved tone of the parts. The handling of traumatic occlusion, however, requires some consideration. Grinding of the occlusal or other surfaces is the usual method of restoring a harmonious action of teeth against their opponents and giving the physiological rest necessary. In extreme cases it is often necessary to open the bite because of excessive overlap in the incisal region. Remember that harmony is needed in what may be called the eccentric occlusion or the occlusion of mastication as well as in the centric or open and shut occlusion. This involves careful study of the compensating curve and the height and inclination of cusps. A very common difficulty is the interference of the cusps of a tipped lower molar with the distal cusps of an upper molar; this may not show itself in the usual or centric occlusion, but let the patient protrude the lower jaw or let either condyle move forward in performing the movements of mastication, and a traumatic occlusion is at once produced. In such an instance the grinding will be more on the lower than on

the upper molar. Deep sulci should be broadened, and sharp cusps should be rounded over to save excessive lateral strain in the movements of mastication.

Grinding for the relief of traumatic occlusion must be done with caution, but must be complete if the desired results are to be obtained. Consciousness on the part of the patient that something is wrong, or complaints of the teeth sliding past each other are indications that grinding has not been carried quite far enough.

In brief, the patient must be able to bring the jaws together and perform the movements of mastication without moving the affected tooth from its natural position in its socket.

Root surgery rests upon certain fundamentals which are seldom appreciated either in the designing or use of instruments. The instrument whether of the plane or the curette type must be not only slender but so shaped as to lie close to the root of the tooth, thereby avoiding distention of the gum tissue. The blade should be centered with the handle to permit of the greatest ease and control in its use. There should be a sufficient variety of shapes to permit placing the blade on the tooth surface at the proper angle without cramping the hand, and with provision for a firm finger rest. The shank must be fairly rigid; a flexible instrument jumps over nodules of calculus instead of removing them. Instruments must be sharp.

The instrument must move over the root surface as evenly and steadily as a tool held in a machine. To accomplish this a finger rest on the tooth, preferably of the same jaw, is essential. The object of root surgery is three fold, to remove foreign material, to remove infected cementum, and to leave the denuded root surface perfectly smooth. The scaler must therefore be not only a cutting instrument, it must be an explorer, taking the place of the eye in determining when the hidden surfaces are properly finished.

These things make up the foundation on which the successful practice of periodontia is built. But back of all this lies one outstanding thought which should be ever in your mind. It is this, the tooth from which a healthy pulp has been removed may have to be extracted for reasons absolutely beyond your control, but the loss of a vital tooth from periodontoclasia is most distinctly an unnecessary evil.

FIXED BRIDGE-WORK.

By H. F. Marston, D. M. D., Minneapolis, Minn.

(Presented at St. Louis May 6, 1919, and Milwaukee June 18, 1919.)

IN PRESENTING the subject of fixed bridge-work for your consideration tonight, please understand that I am doing so with no intention of entering a controversy over the relative values of the two types commonly used. That both fixed and the so-called removable bridges have separate and individual uses, no one will deny; that the extremists of each type have, thru their partisanship, lost to a certain extent their sense of proportion, equally admits of no denial.

Many teeth have been hopelessly ruined thru errors of judgment by the advocates of both systems because of the variable factors entering into the reactions of human tissue. These errors will continue to be made, and the evidence of these errors must and will continue to act as a stimulus for intensive study of bridge-work the world over.

Sane and sound judgment by the impartial observer will alike condemn the fixed bridge man who suspends his span at one end from a lateral incisor, with the other resting upon a tonsil, with the removable fanatic who builds a partial plate to carry the single missing incisor in an otherwise complete dental equipment.

If you will permit me to say so at this time, I believe we are prone to forget the real and underlying cause for bridge-work. Many of us, when considering the course to pursue in a presented case, are thinking too much of the attachment we will use instead of giving equal consideration to the character of the substitute teeth to be supplied.

It is these substitutes that primarily make the bridge a necessity, and it is chiefly with these that we shall deal tonight. However, with your sanction, I would like to take up the preparation and the construction of one method of attachment almost universal in its application.

This is the three-quarter crown, which has come in for considerable discussion the past few years by bridge men everywhere. Condemned by some for reasons wholly unfounded, it has, we might say, weathered the storm stirred up by all innovations, and stands today perhaps the most satisfactory attachment to be made upon vital teeth.

That even the best three-quarter crown we can construct has its evident faults, no one will gainsay; but until our metallographers have carried further their investigations, and we as thinking dentists are further advanced in engineering principles, we can, I believe, find no other form of construction which so nearly fulfills the ideal requirements.

First and foremost, gentlemen, its use absolutely precludes the necessity of pulp devitalization. On the evils of this practice I may add a few words later, and whether you are standing on one side or the other as regards focal infection, I do know you will not dissent when I make the statement that at least the devitalization of abutment teeth lengthens

the time used in bridge construction. Its very obvious advantages, its strength, its appearance—or almost lack of it,—its ease of construction and ease of removal without destruction for purposes of change or repair, are of course familiar to you all and need not be dwelt on here.

The preparation of the tooth to receive this method of attachment varies somewhat in different parts of the country, and, if you who have your own favorite technic will bear with me a few minutes, I will try to describe that method which has been most successful in my hands for the benefit of those who may not be quite so familiar with its details.

Regardless of the tooth to be prepared, please remember that we are dealing also constantly with convex surfaces. Whenever a straight line cut is made thru a convex surface, the end of that cut leaves a distinct and evident angle. Please carry that last statement in your minds thruout the preparation, for the end of the cut is an item of the greatest importance in proper three-quarter construction, as I will attempt to show later on.

After the preliminary studies and impressions have been made, we are ready to begin on the actual grinding of the abutment tooth nearest the median line, selecting this because of mechanical and esthetic reasons and because it is usually the easiest to prepare.

This last is somewhat important because of its effect on the patient. If we have labored hard on a difficult and inaccessible molar for, say, forty-five minutes to an hour, and then, quite exhausted, give the patient the glad tidings that *one* is finished, the patient is quite likely to groan inwardly and think at once how many more of the same dreadful experiences he or she must go thru before the end.

While, by starting at the front, you can usually complete the cutting in from fifteen to twenty minutes, the patient will appear for the next appointment without that feeling of dread so effective on not only himself, but the operator as well.

Using a safe-sided Jo-Dandy disk of about seven-eighths, we began our proximal cut at a point which, when carried straight thru, will run out at that point desired for our gingival margin. Repeat this cut upon the opposite approximal surface, being careful to see that the two straight lines thus produced are slightly —but very slightly—convergent toward the incisal edge. Next, with the three-eighths or half-inch stump stone remove the enamel upon the lingual to about one-half its thickness, this cut to take in only the concave surface of the lingual of the tooth.

Our next cut is made directly across the incisal edge with a very small, sharp-edged mounted point No. 232½. This cut virtually divides the lingual and labial enamel plates, and is carried to a depth governed by the stress to be borne and the incisal thickness of the tooth being prepared.

By all means, where possible, allow this cut when completed to remain a groove and not simply a square-edged shoulder. Care should be exercised in making this cut that the stone does not touch the approximating teeth, and, where necessary, a small fissure bur can be used to finish its ends.

Our next step looks and is simple. But I do not hesitate to tell you gentlemen that its inception was surrounded with considerable profanity and laborious breathing before its simplicity became real.

With the one-half inch concave disks, mounted with the concavity away from the hand-piece, the sharp dividing ridge between the approximal and lingual cuts is rounded off and removed.

Please bear in mind at this stage of the preparation that we have a gingival angle, however faint, outlined upon the approximating surfaces of the tooth and extending partly around to the lingual.

Our next step is to connect the two

pieces of this line by using the mounted point No. 369, and incidentally removing the convexity of the cingulum.

With the tapered cross-cut fissure bur No. 700 contact is made at the incisoapproximal angle, and the groove carried gingivally to that depth deemed necessary, but under no circumstances should the gingival end of this groove result in a definite stop or shoulder. With the next larger size bur the groove is made slightly wider, tho not deeper, and the final cutting and smoothing of the groove is easily accomplished by the use of the mounted point No. 369 ground down to the desired size. With very small stone points round off slightly, any sharp edges and points, and our preparation is complete.

At this point I want to say a few words on the subject of shoulder crowns. In my hands, and in the hands of much more skillful operators than I can ever hope to be, the shoulder crown has not proved the success we hoped and anticipated. Its most glaring fault lay in the physical impossibility of controlling that law of nature which demands that molten gold alloys must undergo a definite shrinkage on cooling. Various means have been tried to insure the resting of the finished casting upon the shoulder of enamel prepared for it, but so far success, to the best of my knowledge, has not been achieved.

It has been my priviledge to examine a large number of both full and three-quarter shoulder crowns upon extracted teeth, crowns made by the most skillful artisans, as well as by those less favored, and I have yet to see a single margin that in its entire length did not disclose at least one and usually many points glaringly faulty.

The shoulder was conceived with but one purpose in view—that of securing a perfectly flush union between tooth structure and metal. That it has failed in its purpose, I believe you will mostly agree. I know I have been guilty—and maybe there are those among you who have also been guilty—of placing too much reliance on that easily used word "burnishing". Considering the inaccessibility of gingival margins and the dense elastic alloys we are using today, I am free to say we move very little metal over the edges of unprotected enamel. (Black on Burnishing.)

Bear in mind, gentlemen, that the shoulder preparation requires deeper cutting than the preparation offered you to-night, and that the formation of that shoulder is made where the enamel is tapering down to its final feathered margin. In most of the shoulder preparations I have examined, the dentin was exposed thruout the gingival area, and wherever faulty fitting of the casting was evident the cement had washed away, leaving a very beautiful ready-made cavity.

In the preparation I offer you, please note that at the end of each vertical cut the natural enamel protective covering is left untouched and in its original thickness and form.

Perhaps some of you will question the strength of the crown so made. In reply, let me state that the strength of the three-quarter crown lies in that bulk of metal which fills the continuous groove, and that the thin sheet of metal passing around the lingual surface serves only to maintain this bulk or metal staple in its proper position. This maintaining of position brings forward the factor of frictional resistance, which is of great importance in proper crown and bridge-work. Near-parallelism of all axial walls is primarily essential if we are to derive the fullest benefit from fixed bridge-work.

Let me give you the simplest form of experiment with this strip of paper and pencil.

Barring caries, a properly made and properly occluded fixed bridge could be worn indefinitely, uncemented, with no other ill effects than that of the unpleas-

ant taste and odor resulting from the fermenting saliva between each tooth and its own partial covering. Cement is resorted to only to seal this minute space, and not to act as a retaining medium.

The preparation given above for the three-quarter crown was obviously that used upon the incisors and cuspids. The preparation of bicuspids and molars varies not at all in principle. Instead of a narrow incising edge, as upon the anterior teeth, we simply have a broader occluding surface to remove, leaving at all times, where permissible, the buccal walls untouched.

In the posterior teeth we sometimes find it necessary to vary our preparation in order to replace tooth structure lost thru decay or accident. Also, we often find teeth rotated and tipped or deflected to such an extent that it is necessary to equally rotate or deflect, in the opposite directions, the preparation. It is not unusual, in cases of tipped lower molars, to groove the lingual and buccal surfaces instead of the mesial and distal.

In tomorrow's clinic I hope to find teeth varying from the ordinary, and will gladly explain, if you are interested, how such attachments can be made with the minimum loss of tooth structure—rotated teeth particularily.

The technic of securing the wax patterns from any of these preparations is so simple I need not take up your time with its details.

In the anterior mouth the matrix is, I believe, unnecessary, while for the bicuspids and molars the Leffingwell and Ivory matrix holders are invaluable.

The open copper bands are almost as efficient as the above-named matrix holders, but somewhat more difficult to separate from the wax. The use of the enameled cloth strips enables us to secure that gingival adaptation of the wax without which our castings would be sorry failures.

Let me impress on you the necessity of securing in your wax pattern that definite line which indicates the end of the cut of the various stones. One-half to one millimeter of excess wax extending beyond this line around the skirt of the pattern, if of tissue paper thinness, because of its elasticity will not interfere in the withdrawal of the pattern from the tooth, and in the metal casting gives us that desired feathered edge which not only compensates for the shrinkage of the metal, but allows us to file and grind to a non-irritating joint and margin.

The casting of your crowns is a subject we will not take up at this time. You have undoubtedly seen clinics where most beautiful results were shown; you have probably listened to and indulged in arguments without end on the relative values of hot and cold flasks, casting at once, and casting the next day, and so forth. If some of you are really desirous of studying the subject as it should be studied, I would advise you to spend some hours with your university teachers of chemistry and physics, with your leading metallurgists and metallographers, and last, but not least, many hours with text-books covering chemistry, physics, metallurgy, and foundry practice. The subject is really limitless, and I regret exceedingly that some fortunately situated member of our profession has not made such study of the subject as its importance demands and has given his findings to the profession.

For your own satisfaction, grind a crude but smooth three-quarter preparation on the end of the handle of an old instrument, make patterns and castings, following the various methods until you can produce a casting that will seat under thumb pressure without grinding or cutting the inside surfaces. When you accomplish that result, do it again and again for proof, and then stick to that method.

Fixed bridge-work is composed of two parts—first attachments, and second,

tooth substitutes or dummies. And, while advocating the three-quarter crown strongly as a most satisfactory attachment in my hands, please remember I do so because of that fact, and not because I for one moment fail to realize that in your hands there are undoubtedly other methods and results of equal merit.

On the subject of natural tooth substitutes, or so-called dummies, I cannot feel so conservative. The old-fashioned facing, ground to surface, with its swaged cusps and body of solder overlapping the buccal surface of the soft tissues, is an abomination in the sight of our profession, and an offense to the taste and smell of the fastidious patient. Its foul collection of food and food ferments would long ago have earned its banishment but for the evident fact that we had nothing to offer in its place.

Years ago we learned that no mouth-used metal, either alloy or pure, no matter how highly polished, could continue either in contact or close proximity with soft tissues and remain clean. Examination of the under surfaces of our so-called hygienic bridges readily discloses that foul, gelatinous mass which is so tenacious as to resist effectually the ordinary brushing, while even upon those surfaces most accessible the disclosing solution reveals the same offensive condition.

These deposits do not adhere to natural tooth surfaces. In the foulest mouth you may seek, I believe you will be unable to locate upon natural teeth that heavy, glue-like mass so common upon the tissue surfaces of old bridgework. Analysis discloses large amounts of epithelial cells, food stuffs in more or less disintegrated state, albumen, fats, and oleic acid.

In experimenting with oleic acid in an effort to learn its effect upon gold, I must admit I was unsuccessful in etching highly polished surfaces of either pure gold or solder as low in carat as eighteen.

Further study along this line could not but be of interest, and very probably would result in findings valuable to the profession.

This gelatinous mass, gentlemen, does not adhere to highly-glazed porcelain, and this fact has enabled us to combine porcelain and gold to secure a bridge dummy that not only fulfills every requirement that did the former types, but has added much to appearance, comfort, and above all else, cleanliness.

With the use of porcelain as mentioned above, we are able to so construct the gingival portions of our dummies that natural dental anatomy is easily secured and almost real cleanliness attained. Shaping the gingival ends of these dummies to conform to that of the underlying soft tissues with which they are in direct and close contact, gives the patient a comfort and sense of oral well-being not heretofore possible.

From the esthetic standpoint this construction is excelled only by the porcelain root in the anterior mouth—which I shall take up presently.

Please allow me to emphasize at this point—for fear of forgetting later—that particularly in the anterior mouth where the extractions have not been recent it is possible, by the use of this individual porcelain saddle, to so imitate nature that more than once have members of our own profession failed to locate the dummy restoration until very careful scrutiny has been made. Why these individual saddles stay clean, not only to the eye, but to the disclosing solution as well, I will explain when discussing the porcelain root.

Glazed porcelain is the only substance used in dentistry today that is tolerated by soft tissues without irritation.

This established fact enables us to fill, or nearly fill, that opening left in the process following extraction by a substitute structure which, because of its non-irritating properties, is kindly received and permanently adopted by the surrounding mucous membrane.

By no means do I wish you to understand that there is a union between the membrane and porcelain of any sort. This is no implantation idea, revamped and made over for your entertainment. Again I say, there is no union of any description; but there *is* an adaptation of the soft tissues, so close, so accurate, and so extremely snug, that the introduction along the surfaces of a very fine, straight explorer is attended with considerable difficulty, and in some cases is impossible without actual penetration of the tissues by the instrument.

I would like to say at this point that the only differences in construction between the individual saddled dummies I spoke of above and the dummies carrying the extended porcelain roots lies in the fitting of the gingival end to soft tissues in the first, and the fitting of its circumference and length to a lined, bony socket in the second.

In explaining the construction used in our porcelain root cases, the same technic applies for the saddle cases up to that point where fitting of the biscuited portion begins; and, as the root cases are probably of most interest to you, I will take up as rapidly as possible the construction of a simple anterior bridge carrying one or more porcelain roots.

At the first sitting, as a preliminary step, an accurate impression is taken in compound of that group of teeth which is to constitute our completed bridge, as well as radiographs of roots of teeth to be replaced. The shade is also selected at this time and marked on the tray, and this is laid aside for future use.

The preparation of abutments is then begun, as explained in the earlier part of our paper. Wax patterns are obtained at once of one or more of the teeth just prepared, and the patient is dismissed until another time.

Let me emphasize the importance of always taking patterns immediately after the preparation. No two preparations are ever just the same, and much better results are obtainable if we do this work while the individuality of that one tooth is fresh in our minds.

When all wax patterns are complete —I keep mine in groups and cast as such in the same flask—the patient is dismissed until the next appointment, with instructions to be somewhat careful of the sharp incisal edges. In this interim we will pour the impression obtained, and from this select the facings to be used according to the type and color desired. Upon these facings we now build porcelain roots, over-sized, and bake them to what you all know as the biscuit bake.

I should state here that those who may wish to have the laboratories do their baking for them should furnish the laboratory with the impression and shade immediately after they are secured. In this way you are able to operate at the same time on several pieces of work in course of construction.

At the next appointment our castings are fitted to place and partly polished. A compound impression of the full upper and lower is then taken, care being exercised that the impression is extremely accurate as regards gingival outline of all teeth entering into the bridge. The patient is then sent to the oxodontist, who is instructed to return the patient with the extracted teeth to you as soon as possible. Your exodontist will pack these sockets with iodoform gauze if you so request, but personally I much prefer and advise that they be left open.

The patient is then given instruction as to the toilet of the mouth, and dismissed for approximately three days. The cast attachments are removed before dismissal and placed in the impression.

We now take the extracted teeth, and with the engine stones reduce the length of the roots by about one-third to one-half their length, and grind away to a depth of approximately one millimeter the entire circumferential area. This

grinding is carried sharply up to the gingival line. These teeth with the reduced roots are then carefully inserted into the impression, securely waxed to place, the root surfaces coated with vaseline or oil, and the model poured with either S. S. White's Sump or some similar investment. Mount your models as made upon your articulator with the proper bite, and you now have an exact copy of the natural dentures with all teeth in place. Remove the extracted teeth from the model with extreme care, so that the gingival margins of your model are not fractured.

Now take up your biscuited roots, and with engine stones grind to fit the sockets in the model, starting with coarse stones to remove the greater excess, and finish up with the fine stones and sandpaper disks.

The fit of the root to its socket is unimportant except at the gingival region, where care should be taken that the root fits accurately and closely. (Tight fit because of elasticity of free margin.)

The roots are then thoroly scrubbed with brush, soap, and water, dried, and returned to the furnace for the fuse or final bake. This bake should be carried to a high glaze—somewhat beyond that used in porcelain jacket or inlay work.

When cool, the facings are now beveled at incisal and approximal edges, the gingival end of the root being ground to conform to the lingual line, and all waxed to place.

The plaster guide-block to preserve the set-up is then poured on the labial surfaces, and when hard is removed. The facings are now cleansed with chloroform to remove the black wax, and ground for the reception of such fillings, if any, as existed in the natural extracted teeth. The reproduction of both approximal and L-fillings in cast dummies of this type is so simple and so pleasing, I would respectfully urge that you do more of it than is perhaps your usual custom.

Returning the guide-block and facings to place, oil the pin side of facings and press firmly to place the water-softened casting wax. Have your wax sufficiently hot so that you will be able to force it into all the angles and crevices, including the filling cavities mentioned above. Carve the wax to suit, being particularly careful to reproduce the cingulum and other characteristic markings upon the lingual surfaces of the extracted teeth, which is so necessary for proper excursion of food and the consequent massage of the tissues, with its attendant benefit.

We will then remove our entire block of facings with wax backing in one piece, removing the facings with the aid of sticky wax and sprues, fill the pin holes with carbon points, invest, and cast.

For the sake of cleanliness, and to dissolve the oil film from our facings, keep them in a small bottle of chloroform until ready to fit to the cast backing. With our casting made, we remove the carbon points, either with the engine burs or with hydrofluoric acid, clean thoroly and carefully fit the facings to their respective places.

Extreme care should be used at this time not to fracture any sharp edges of our porcelains.

Due to shrinkage in our large bulk of metal, it is usually necessary to relieve the approximal surfaces of the porcelains in order that they may be not only seated properly, but that they be not quite in contact in the completed bridge.

When all facings are seated, the block is returned to place on the model, the guide-block fitted for test, and the casting waxed to position. The guide-block is then removed and the labial portion of the model cut away over each of the roots. The facings, with their roots, are then easily removed from the casting; the waxing between attachments and dummy casting completed, and the case invested and soldered.

Our bridge is now ready for the try-in. Carefully cleanse the abutment teeth

with dioxygen and warm water, and apply Lugal's solution to socket linings. The metal frame is then carried to place to verify our impression and soldering operations, as well as the occlusion. Remove frame and, selecting a facing, coat the root lightly with vaseline, introducing gently into the socket with a rotary motion, coupled with steady but continued pressure. Imprisoned air may be released by the introduction of the straight-pointed explorer and a rocking motion applied to the root. When soft tissues have been expanded enough to allow each porcelain root to be introduced to its proper depth, the frame and facings are cleansed with chloroform, the facings cemented to place, and as soon as cement has hardened sufficiently the entire bridge is pressed gently and slowly to position.

It is my custom to require my patients to wear a bridge uncemented for from two or three days to a week or two for two reasons: first, I can in this way watch developments within the sockets—particularily at the apex where infection has been present; and second, because it allows me to secure a more perfect occlusion than is possible where this occlusion is established while the patient is in the chair, moving the jaw at command. The voluntary and involuntary movements of the mandible are proportioned approximately twenty-five and seventy-five percent respectively. Actually, a patient is able to exert but few—very few—of the common masticatory motions with the mouth empty and the mind attempting to direct the various masticatory muscles.

You all have had occasions where you have ground and polished a filling, crown, or bridge until you felt certain proper clearance was established thruout, only to have the patient return in a day or two with one or more points indicating excessive pounding. In case it is necessary to hasten the final cementing and occlusion of a bridge, I find common chewing-gum a decided aid. Instruct the patient to walk around the hall or the building for a few minutes, vigorously chewing one or two pieces of gum, and you can in this way secure a pretty close estimate of the occlusion to which the new piece of work will be subjected.

Now as to our sockets and the porcelain roots introduced permanently into them. Some of you perhaps are wondering just what has occurred and is occurring. Examine the margin of our extraction sockets twenty-four hours after the teeth have been removed, and you will discover an invagination of the surrounding epithelium has taken place to a depth from, say, two millimeters to five or six millimeters, depending on the age and physical condition of the patient. Diabetics are notoriously slow healers, so, in case you do not already know the clinical history of your patient and the sockets fail to indicate normal activity, inquire as to conditions or presence of this disorder. Two particular cases I have in mind where this condition was known to exist, and in both it was necessary to wear the bridge alternate days before sufficient tissue activity had taken place to insure a permanent healing.

This invagination continues rapidly, covering over the newly organized apical tissue, until in the patient of middle life we have the socket completely lined in from four to eight days.

Where the porcelain root is used, this invagination of tissue closely adapts itself to the glazed surface, and by the stimulation afforded rapidly approaches that condition normally indicated by mucous membrane in the gingival region. Bone growth begins to fill the apex of the cavity as soon as all infection has been dissipated, and this bone growth continues until it has approached within approximately one and one-half to two millimeters of the porcelain root. It is here apparently stopped by the soft tissue surrounding the root and remains sta-

tionary. This soft tissue about the root we have termed, for the sake of easy designation, the false peridental membrane.

Macroscopic examination of these sockets of all ages up to more than four years after insertion reveals a firm, moist, pale-pink surface, with every indication of perfect health.

Why it is so and why it stays so is really simple. We have a cone-shaped cavity, with one end open; fitting this cavity closely is a highly glazed, smooth-surfaced impermeable mass, which moves in and out a minute distance whenever pressure is exerted upon the occluding surfaces of the bridge structure of which it is a part. This pumping motion, coupled with the normal exudate from the lining of the soft tissue, creates a constant tho very slow flow toward the gingival outlet. This pumping motion, under proper conditions, constitutes a massage which is mildly stimulating, but not irritating. Mind you, gentlemen, I say under *proper conditions.* You can readily see that if loose abutments are used, if our porcelain roots are not smooth, or if our occlusion is abnormal, irritation results and this is followed by tissue destruction.

Please allow me to say here that one of the commonest forms of irritation found in our earlier bridges of this type was due to poor technic or careless workmanship. I refer to the actual contact upon the bony walls of sockets by poorly shaped or poorly placed porcelain roots. With the methods I have tried to outline tonight none but a blunderer can come to grief.

I have taken much of your time, gentlemen, in attempting to outline but two types—really but one with a modification —of fixed bridge-work that is in general use in the Northwest. I present it to you, —not as an experiment—but as a type which has been tried and proved successful. That it will so prove in your capable hands is easily attested by the reputation St. Louis dentists enjoy wherever the profession is known.

There are many, many phases of this subject I would enjoy discussing with you, but your patience, I know, must be about exhausted.

I would like to tell you how we make two—and three— tooth anterior bridges directly in the mouth in two appointments.

I would like to tell you the methods we use to reproduce the various characteristics of the natural teeth, their enamel flaws, their erosion, fillings, blemishes.

I would like to explain how easy it is to repair a broken porcelain root or facing and how we remove bridges constructed of three-quarter crowns without destroying them.

I would like to take you to the laboratory work-bench and go over the little details that we all do from day to day, and yet all do just a little differently from our neighbor.

I would like to talk focal infection with you—or, rather, I would like to hear your views. I am not competent to discuss it—I only know what I have seen and do see from day to day at the hands of that man beloved in St. Louis as well as thruout the Northwest, Dr. Fred B. Kremer.

I would like to tell you how Dr. Monson, of St. Paul, has demonstrated the connection between loss of hearing and malocclusion, and of some of the results he is obtaining.

I would like to spend hours in the offices of every one of you, for it is in these little private sessions we make friendships that are so often prolific of good, both professional and social.

I would like to tell you about a dental laboratory in our city of whose owner it can be said and is said, "No other one man in the Northwest has done so much for the advancement of modern dentistry."

THE REMOVAL OR RETENTION OF PULPLESS TEETH.

By Joseph A. Pollia, M. D.; Walter C. Alvarez, M. D.; Frank L. Platt, D. D. S.; Ernest D. Chipman, M. D.; Professor Karl F. Meyer; Robert Burns, Jr., D. D. S., and Guy S. Millberry, D. D. S., With an Epilogue by A. F. Lucchetti, President of the San Francisco District Dental Society.

(A Symposium presented before the San Francisco District Dental Society, and the San Francisco County Medical Society.)

Jos. A. Pollia, M. D.

GENTLEMEN: This question of devitalizing teeth is an old one, and it is not going to be decided tonight. But what I would like to do tonight is to set you thinking, and whatever stand you take as individuals, you will have your own thoughts, to sustain your views. To the gentlemen who would like to save the devitalized teeth, I would like to make an explanation. By the term "devitalized" I mean a tooth which has not only had its pulp removed but one in which the pulp has died from trauma, or from a filling too close to the pulp, or from any other cause. And to these gentlemen who would save the devitalized teeth, I would like to ask a few questions.

Have these gentlemen ever considered the pathological relationship between a devitalized tooth and the alveolar process? Have you ever considered that the sterilization of a closed cavity, that has been infected, is a well nigh practical impossibility? Have you as individuals, made a keen, unbiased resume of radiographic findings, and does the report of prominent clinicians cut any figure at all in this discussion? Now it is the pathologic relationship: it is this subject of sterilization: it is the radiographic findings, and the clinical data. These are the four points I would like to put into your minds for you to think over and over, and whatever your decision is, you have some basis upon which to make it.

Now what is the pathologic relationship between devitalized tooth and the alveolar process? A devitalized tooth has some circulation. Of course if it did not, nature would throw it off. How much circulation it has is a matter of discussion, but you will all admit it is considerably less than that of the normal tooth. A tooth is composed of highly specialized types of tissue, but we are not interested in the tooth: we are interested in the bones that surround it. What are these bones that surround it? Are they made up of highly specialized osseous tissue? No. Therefore, no one has the right to believe as many dentists claim—in fact if not in word—that the bones of the jaw are not subject to the same pathologic laws as are other bones in the body under the same conditions. Now, what are these pathologic changes?

When an area of diminished circulation occurs in the body, this area becomes prone to bacterial attack. This is true of any other structure. A devitalized tooth, as we have just shown, is an area of diminished circulation, and if you have

not infected it in the process of your devitalization it soon becomes a prey to bacterial attack, and by reason of its high specialization it may resist the end results of bacterial infection, but the intimately related bones of the jaw do not. Therefore you have that line of changes described by the radiogram extending from the slight peri-alveolar thickening down to the enormous areas you have had experience with. Now you may say, Nature is throwing off a protective wall: that this area of thickened bone which I called sclerotic bone a year ago is a protective wall, but in the process of my studies all those areas which were large and plainly discernible in the radiogram, and which all men admitted were pathological, I cultured mixed infections, with the staphylococcus, predominating, and where we had the sclerotic bone described I cultured streptococcus in most cases, and the diplococcus of pneumonia in a few. So therefore the large so-called abscessed area is in reality due to the digestive property of the staphylococcus, and the sclerotic area is not the protective wall but is due to the properties of the streptococcus. Therefore this "abscessed area in one place, and the sclerotic bone in the other, is a tissue reaction to bacterial properties and not a defensive expression.

This subject of sterilization has occupied a large place in the conservation of pulpless teeth. Do you realize that for many years surgical science has been looking for that antiseptic which will do to wounds what you claim to have been doing in infected teeth? The perfect antiseptic,—the one which will kill bacteria and not affect the surrounding tissue. We have found no such substance as yet that has this selective property. However, the Rockefeller Institute has recently demonstrated that trauma is one of the most important etiological factors in the production of infections, and you can produce trauma just as effectually with a strong chemical as with a blow of a hammer. Therefore in using a chemical which is strong enough to kill the bacteria you have also killed the tissue cells in the immediate vicinity, and furthermore you have injured those beyond. In other words, if yo do not destroy the infection at that particular sitting the traumatized tissue has lessened resistance, reinfection occurs and you are just continuing the process you are trying to eradicate.

Of all the diagnostic methods in dentistry, if there is one that condemns the pulpless tooth without the slightest reserve, radiographic is that one. I have heard many prominent surgeons say the radiograph never magnifies. I agree, but would add that it never detracts. A radiograph properly taken will show with mathematical exactness pathological changes which have taken place. Gentlemen, I have seen in the radiogram such minute pulp changes as would take place in the place of a small filling in a tooth, no matter how far away from the pulp. And we might ask furthermore, what does the rediogram show in these cases of sterilization? In many hundreds of them, which I have seen, one—just a single, solitary one where regeneration occurred, and I never in the wildest dreams of my imagination would call that bone normal. And of devitalized teeth I have never seen one out of many thousands of radiograms, not one with a normal apex, and, gentlemen, I am sorry that I have not the time this evening to show you that wonderful picture of apical change starting from the delicate thickening of the peri-alveolar layer of bone and running up to the most common clearly defined eroded area with which you are familiar.

I would like to say another thing—as to clinical data. Clinical data is unanimously in favor of removal. That means entire removal: thoro removal. It does not mean leave some in and take some out. It means take it all out that has the slightest bearing on the case. There are cases where the teeth have been taken out which looked bad in the radiogram,

and those which showed smaller changes were left in. A majority of patients receive no result until you take out the innocent appearing members. You may say, Doctor what about this case—where they take the teeth all out and get no result? In such cases I would say the method of removal is a fault, and I am pretty sure if the job were done surgically clean (there is a big difference between clean and surgically clean) that you would get your result. Furthermore, I have had the good fortune to tabulate 125 cases where focal infection existed in the teeth, and each and every one that went to operation and had the thoro, clean surgical operation done, everyone at least was much improved, if not entirely cured, and I would like to add, aside from that, that that is more than you can say for any other one medical idea.

Now if I have raised in your minds pathological, bacteriological, radiographic and clinical objections to the devitalized tooth I think it has been a pretty good evening's work. I want to thank you for your kind attention.

Walter C. Alvarez, M. D.

Day after day I see people who have had a half dozen or more teeth extracted. Their former physicians had promised them great things; in some cases had even guaranteed a cure, but here they are still suffering and now greatly discouraged. Many have no chewing surface left, and the remaining teeth are often so distributed that the only thing to do is to remove them and put in plates. To my mind one of the saddest features is that, in many of these cases, an experienced physician might have foretold the unsatisfactory outcome and might have warned the orthopedist or the dentist to proceed cautiously and conservatively. The arthritis may plainly have been gouty or tubercular; the headaches may have been due to nephritis or to advanced arteriosclerosis, and the pains in the shoulders may have been due to degenerative changes in the aortic arch. Sometimes I have secured the radiographs which were used in deciding which teeth were to come out and have been unable to find more than one or two roots which after years of experience I would call infected. In some, downward projections of the antrum had evidently been mistaken for abscesses. In others, it seemed to me that the physician, quite oblivious to any possible value of the teeth to their owner, must have ordered their extraction simply because he believed it a panacea for most diseases.

I believe we have lost our heads over this thing and that the time has come to call a halt. Men have gotten such beautiful results in some cases by extracting teeth that some of them are now trying to explain most diseases on the basis of these focal infections. In practice, they pull the teeth first, and if the patient returns unbenefited, they can then look to see what is the matter with him.

As the enthusiast often regards the conservative as a man who must be ignorant of the wonderful results which can be gained by using his methods, I wish to say that since 1911 it has been practically a routine in my office to radiograph all suspicious teeth, and I have had hundreds extracted. I would emphasize the fact that I have seen my share of the miracles described by my radical friends—I too have seen inflamed joints go down over night; so-called tuberculous glands disappear as suddenly; headaches leave for good, and so on; but these things have not blinded me to the fact that for one miracle I have seen many failures and disappointments. The radicals who from their writings would seem never to have had any failures may perhaps ascribe my unfortunate results to conservatism and to the choice of poor dental surgeons. Altho this argument may apply in regard to those cases in which I ordered the extractions, it can hardly apply to those people who were "cured" by these same radicals

but who, after a period of relief are again going the rounds of doctors' offices.

We must recognize these failures; we must face them squarely and study them with particular care. Many people are not benefited by the extraction of teeth no matter how thoroly it is done, and no good can come from further refinements of that technic. In the middle ages if a man died it was because he had not been bled enough, or because it had not been done from the right vein. Today we know that bleeding is useful in only a few conditions. Similarly, the day will come when focal infections will not be dragged in to explain all ills of the flesh. Seeing that there are these possibilities of failure we must be more honest and conservative with those who put their lives in our hands. By all means let us continue to look for alveolar abscesses but, if we are to keep the respect and confidence of the public, if we are to avoid damage suits, we must be more careful what we promise some of our patients in return for a toothless mouth. Before deciding whether any or all of the suspicious teeth are to come out, the physician must look the individual over from head to foot. If he is young and sound, and if he has a dangerous arthritis, endocarditis or severe headaches for which no other cause can be found, I believe we are justified in insisting on a thoro removal of the diseased tissues. When, however, the patient is old and failing; or when we find high blood pressure, arteriosclerosis and nephritis, let us be careful. Focal infections may perhaps contribute to the development of these chronic degenerative diseases, but I feel sure that they are not the only or even the principal cause. Certainly my experience has been that altho the removal of the teeth will sometimes seem to give the patient a new lease of life, the arteries continue to harden, the pressure continues to rise, and sooner or later the symptoms return. Hence it is that in these cases we should carefully consider all the factors involved. If the infected teeth are not serviceable, if they are loosened, perhaps hanging to wobbly bridges; or if their crowns are gone, there need be no question as to their removal. But if they are strong and serviceable, if the areas of rarefaction are small and questionable, and particularly if restoration by bridges will be impossible, let us be frank with the patient. Let us tell him that the proposed extraction is more or less of an experiment; that we will not promise him anything; but if his troubles are annoying him so much that he wants to leave no stone unturned in his treatment, he can try it.

It is one thing to order a dozen extractions when a man lies crippled with an acute or chronic arthritis, and another to order them because he feels nervous, has no energy, has indigestion or pimples on his chin. In the first place the patient is desperate, he is willing to take any chance to get well and will more readily forgive mistakes that arise thru overzealous efforts than mistakes arising thru lack of interest and neglect. In the second case, he is in no mood for big sacrifices; the treatment, even if successful, may cause more annoyance than the disease, and the physician may come in for a great deal of blame.

We must be careful, also, what we promise to those who, in addition to their root abscesses have other sources of infection which cannot be so easily removed. I refer particularly to chronic bronchitis, sinusitis, prostratitis and urinary infections, all of which may be persist in spite of expert treatment. We may perhaps have trouble, too, on account of pelvic inflammation and gallbladder disease.

The radical group of dentists tell us that most of our failures to cure are due to the fact that we have not removed all the diseased tissue from the jaws. They would like to open up the jaw widely and dissect away the alveolar process until the tooth can almost drop out of itself. Naturally these men have a certain amount of right on their side. I too have

seen a number of cases in which diseased bone or the abscess itself was left by the man who extracted the teeth. There are some people in whom the X-ray shows a wide-spread change in the bone around the diseased teeth, and in these cases I think it is probably advisable to remove enough of the inner or outer plate so that the operator can see what he is doing. I feel sure, however, that in the average case, a good extractor can remove the abscess, sometimes entire and hanging to the apex of the tooth.

Many of our patients will return still complaining of their old troubles even when the X-ray shows that the removal of diseased tissue has been complete. In some cases the bacteria which may originally have entered thru the jaws seem to have gotten so firm a hold on the joints, the heart valves and other tissues that they will not leave simply because their old port of entry has been closed. In other cases, irreparable damage has been done and the joints cannot return to normal even after the disappearance of the infection. We must remember, moreover, that focal infection is probably not the only cause of arthritis and our clinical study of this condition will not always begin and end with the search for pus.

I believe the *main* reason for our disappointments is that in many cases the alveolar abscesses seem to exercise no demonstrable influence on the patient's health. Time and again I have seen powerful, healthy-looking men with large alveolar abscesses which they had carried for most of their lives. They maintained that they had never had a headache or a twinge of rheumatism and in fact had never needed a physician. When such a man remains disabled after an injury we often try to rehabilitate him by removing the abscesses. Naturally, we often fail, simply because we take away something which has never had anything to do with the case. It may be, sometimes, that these sacs are as much outside the body as is a calcified tuberculous gland in the lung. When further bacteriological work has been done we may find that some of them are sterile, the bacteria having died out just as they do in pus-tubes. Particularly in the case of the smaller areas, the necrosis may never have been infectious in origin but may have been produced by the chemicals used during the preparation of the root-canals.

Many of the dentists have become so frightened over the terrible results which they think must follow every root infection that they are refusing to fill any root-canals at all. They feel that the risk to life and health is so great that a man should immediately sacrifice every dead tooth in his head. Certainly the thousands of people who for the last thirty or forty years have been chewing contentedly on dead teeth (without signs of root infection) should be grateful that these radical ideas did not prevail when they were young. The trouble with many of our dentists today is that they do not know enough about the wonderful defenses of the body against bacteria. These defenses are particularly efficient in the mouth where in spite of the rich flora and the continual trauma, wounds heal with surprising rapidity. Bacteria are constantly getting thru the first line of defense only to be stopped at the second, and I see no reason why the body cannot in many cases protect itself perfectly from the activities of a few invaders which have reached the apex of a tooth.

To conclude: In view of the fact that the most thoro removal of focal infections often fails to cure arthritis and other diseases, let us be more honest and conservative with our patients. Let us be careful what we promise them. Let us save serviceable teeth whenever possible. Above all, let us do unto our patients only what we would have done unto ourselves if their teeth were in our heads.

Frank L. Platt, D. D. S.

A few weeks ago I received a letter from one of the best known and best loved dentists in America, Dr. C. N. Johnson of Chicago, in which he stated he had advised one of his patients to consult me regarding her teeth, as she had been told that one or more of them must be extracted or she would be liable to be afflicted with endocarditis, neuritis or some other equally awful complaint. He asked me to treat the young lady gently both physically and financially as she was much alarmed about her condition and not in a position to pay a large fee.

On the same day this letter was received someone called up my office and asked for one, the best known "dental surgeons" in San Francisco; (I wish our colleges would tell us why the degree of D. D. S. makes one man a "dental surgeon" and another "only a dentist") on being told that Dr. Platts office had been called the person at the other end of the line said "Oh, I meant Dr. Platt", and then asked for an appointment. It's odd how names will get mixed up some times even when there is a sharp distinction between them. On arriving at my office the patient proved to be the one to whom Dr. Johnson had referred and on examination I found the lower right first molar practically floating in a nice little sea of pus, the outcome of a chronic alveolar abscess.

I advised the extraction of the tooth, told the patient, who was greatly alarmed at the prospect of going to a hospital for the operation, that the tooth could be removed in my office with little discomfort and that she was not in very immediate danger of a fatal termination of the affair.

I wrote Dr. Johnson that I had advised the extraction of the tooth and told him of the condition in which I found it, that one of my specialties was tempering the wind to the shorn lambs, particularly the ewe lambs, and that I would treat his patient as gently as possible in every way. Then I added this postscript "May I not say," as Woodrow so aptly puts it, that in extracting a tooth I do not think it always necessary to remove all the osseous tissue below the eyebrows.

I received an immediate reply from the doctor thanking me for attending to his patient but saying he thought I must be very unorthodox indeed if, in extracting a tooth, I left any of the osseous tissues between "the clavicle and the eyebrows." I extracted the tooth in question under local anesthesia, moderately curetted the abscessed area and am happy to state, to use our stereotyped formula, that the patient made "an uneventful recovery."

The sad fact remains however that I appear before you this evening an unorthodox and conservative member of our profession who still believes that some pulpless teeth may and should be saved. When Dr. Milliken first asked me to prepare a paper for this evening he mentioned a subject which he thought would be suitable for the occasion; a few days later he gave me another subject and then another and yet another, until I was somewhat at a loss as to just what to write about until it occurred to me that possibly he thought that in a multiplicity of subjects there might be one which would fit the paper I was to write, and I wish to assure him that I fully appreciate his thoughtful kindness and hope his intuition has not led him astray.

To get down to the subject of the evening which according to the latest advices at hand, is, "The Retention of Pulpless Teeth," let us start with the proposition that the extraction of a pulpless tooth is a tacit admission of failure on the part of dental science to maintain in the mouth an organ placed there for a number of important purposes and the loss of which cannot be wholly repaired.

Of course this applies only in a general way for many factors enter into the

case, such as accident or injury or personal neglect on the part of the patient.

Under ideal conditions it would never be necessary to devitalize a pulp nor would pulps ever die as a result of dental caries, the irritation of filling materials, thermal shock, bacterial invasion or any one of the many causes entering into pulp degeneracy. But ideals are seldom attainable and under present conditions devitalization is a very real and ever present problem with which we have to reconcile our views.

The present trend of dental education tends largely toward the prevention of disease and much good is being accomplished by the study and development of dental prophylaxis, not alone as it applies to office procedure but also as its importance is being impressed on the public mind thru instruction in our infirmaries and public schools and in the columns of the press. Keeping pace with this educational progress and in some cases seeming to actually outdistance it there also has developed a very general interest in the relationship of dental disease to systemic disorders and it is in this relation that the problem of devitalization in its many aspects is mostly concerned, the term devitalization being used in its broadest sense as applying to all those conditions arising from the loss of vitality in the dental pulp.

We seem to be pretty well agreed that aside from those dental diseases which concern the gingiva and peridental membrane, those most directly associated with systemic disturbances arise from pulpless teeth.

It is assumed in this discussion of course that the relationship between dental and systemic disease is accepted as an established fact.

The crux of the whole situation then is, admitting that those dental diseases arising from devitalization are largely responsible for systemic disturbances, shall all pulpless teeth be extracted, or shall an attempt be made to render them inert so far as their ability to incite disease is concerned or to cure the purely dental diseases incident to devitalization, leaving the teeth in situ? A review of dental literature reveals the fact that a very large proportion of our contributors are interested in the problems related to the sterilization and filling of root-canals and to the cure of alveolar abscesses, while the names of those who advocate the extraction of all devitalized teeth are conspicious by their absence from the list of those who are known as students and investigators of dental problems.

That pulpless teeth may be sterilized and safely retained in the mouth, and that alveolar abscesses may be cured seems evident from the published reports of investigations carried out by many of the best known members of the dental profession.

In the Dental Cosmos for April, 1917, page 393 Dr. Herman Prinz, in an article on the "Electro-Sterilization of Root-canals" states "The surface of an infected root-canal of a tooth may be completely sterilized to the depth of a few millimeters by a weak direct current in the presence of a one percent sodium chlorid solution."

Dr. Percy R. Howe in an article entitled "A Method of Sterilizing, and at the Same Time Impregnating with A Metal, Affected Dentinal Tissue", on page 891 in the Dental Cosmos for September 1917, states "By this method it is possible to completely sterilize not only a putrescent pulp without removing it, but also the dentinal structure of the root as well." Also "We have found it effective in the treatment of chronic abscesses."

In The Journal of the National Dental Association for April, 1919, page 345, Dr. Black states: "I reported the examination of radiographs of some 1500 root-canal fillings, which showed about nine per cent of failures in those cases in which the fillings appeared to have been well made."

In the Dental Cosmos for December 1918, Dr. Prinz reports the use of

dichloramin-T in an article entitled: "Sterilization of Root-Canals" and says "Experimentally, proof can now be furnished that infected apical areas and even granulomas, may be completely eradicated by the application of dichloramin-T solution, provided that the solution is brought into intimate contact with the infected zone."

Dr. Price of the Research Institute of the National Dental Association reports that the silver precipitation method advocated by Dr. Howe and the application of Iodin and Formalin will each positively sterilize pulp canals and dentin.

Numerous authors report success in the amputation of roots and the resection of root apices, among them being Drs. Nodine and Hartzell.

I have quoted but a few of the many articles on the treatment of pulpless teeth appearing in our journals during the past three or four years, there are many others and I find none advocating the extraction of all pulpless teeth.

It is a matter of common experience with many of us to find in the mouths of our patients pulpless teeth which have given from five to fifteen or more years of comfortable service with no history of systemic disturbance which by any stretch of the imagination could be attributed to their condition or retention.

It is also true that many cases are called to our attention where the extraction of such teeth has failed to cure the systemic disease of which they were supposed to be the cause. Many of us can also call to mind the apparent cure of or temporary relief from, various systemic lesions by the extraction of pulpless teeth.

So far as I am aware there is no way to positively determine whether one or more pulpless teeth in the mouth of a patient may or may not be the cause of systemic disease with which he may be afflicted except by the extraction of the teeth, a rather heroic experiment, not altogether justified by a more or less problematical diagnosis.

Bearing in mind the fact that the replacement of lost teeth involves to a very great extent the health, vitality, appearance and more or less mutilation of the remaining teeth in the mouth, in the case of partial restorations, and that artificial substitutes ranging from fixed bridge-work to full dentures are said to be from fifteen to sixty-five per cent less efficient than natural teeth it seems to me that those who advocate the removal of all pulpless teeth are assuming a responsibility not altogether warranted by our present knowledge of the part played by such teeth as active agents in causing or promoting systemic disturbances.

We may agree I think that alveolar abscess either acute or chronic is not a condition tending to promote either the health or comfort of any one, neither is the presence of pus anywhere in the body looked upon as a desirable state of affairs, but is it not possible in dental as well as in general surgery to cure such a condition without resorting to the removal of the organ or part involved? Is it not possible to cure an alveolar abscess?

Look thru the pages of our journals for the past three or four years or even more, and you will find the exponents of ionization, root resection and many other forms of treatment of pulpless and abscessed teeth all reporting cures and supporting their contention with radiograms showing the reorganization of tissue in diseased areas.

These reports are made by men of national and international repute as careful investigators of the subjects of which they write; are such reports to be ignored and all pulpless teeth extracted because some of them may possibly cause systemic disease or be beyond the power of anyone to save?

Are such teeth not only to be extracted but surgically removed together with all the super-adjacent tissue which can be reached with a curette or mallet and chisels?

I contend that in our present knowl-

edge of the *direct* relationship between dental and systemic disease we are not justified in resorting to such radical measures. I do not believe we are justified in permantly crippling and disfiguring our patients excepting as a last resort when all our efforts to cure by other means have failed.

Not so very long ago a prominent oralogist, a member of this society, told us, before long we would be compelled by law to extract all pulpless teeth, and quite recently another radical friend of mine told me it would shortly be necessary to secure the written consent of the patient before resorting to devitalization. These prophesies may come true but at present I am constrained to think them but the peculiar vagaries of imaginations much overwrought by seeing in radiograms the things which are not there. They are but the dreams of enthusiasts, remembering of course the definition of a dream which the husband gave his wife when she said to him: "Before we were married, Henry, you said I was a dream," and he replied: "Yep, old woman, and I say so yet, a dream bein' a thing you wake up from and find aint so."

We are told quite often of "instantaneous reactions" and "marvelous cures," the result of the extraction of pulpless teeth, and yet when we meet the patients and congratulate them on their recovery and they look us coldly in the eye and tell us in perfectly intelligible language that they are not a profane bit better than before the operation are we not justified in thinking that imagination plays quite a prominent part in these wonderful diagnoses, operations and cures?

This craze, this peculiar epidemic of surgical exodontia, is not, however, without its compensations.

It may be necessary in some cases and few will count or particularly care how many thousands of dental guinea pigs are sacrificed on the altar of unscientific research before the pendulum of reason will vibrate with even beat and we will know when to extract and when to save.

And what an opportunity it affords for the dignification of the often simple and somewhat plebian operation of tooth extraction! dignified to the realms of hospital procedure, at so much per dig, to the great gratification of the patient who would join Irvin S. Cobb in "Speaking of Operations" and the emolument of the operator who rates the worth of his services more by the fee he can command than by their actual value and necessity to his patient.

Ernest D. Chipman, M. D.

The specific question as to whether so-called devitalized teeth are actually dead teeth and as to what percentage of dead or devitalized teeth are potential foci of infection, are of interest to the physician but their solution rests with the dentist.

The definite relationship between many systemic disorders and tooth infection brings the dental and medical professions into much closer contact than was dreamed of a decade or two ago. Personal experience in seeking the role played by focal infection in the etiology of certain skin diseases has not only interested me particularly in the question of tooth infection but has impressed me deeply with the necessity for some common basis of understanding between dentists and physicians as to how cases, in which both are concerned, should be treated. It is with reluctance that I confess to many disappointments in my relations with dental confreres. I am not unmindful of the fact that possibly they also have felt disappointment in their relations with medical consultants. If to the physicians it has seemed that the dentist has temporized with teeth which were harboring death dealing charges of infection, perhaps also to the dentist it has seemed the physician has too hastily demanded the removal of some fine reconstructive work which has been built up painstakingly and which

the dentist feels has no bearing upon the condition which the physician is treating. These remarks are offered then with the thought of attempting in some measure to establish a closer community of interest and to help each to approach the problems of mutual concern from more nearly the same point of view.

This common view point is a paramount necessity once it is conceded that oral sepsis is the starting point of such morbid conditions as rheumatism, neuritis, endocarditis, erythema nodosum and zoster—to mention only a few of the diseases of which focal infection is considered the cause. If one says that everyone concedes these points, a denial must be entered. It is by no means uncommon to hear both dentists and physicians say, "There is probably something in it but it is carried too far." This is unfair, for if there is anything in it at all there is enough for each demonstrable focus of infection to be removed. Moreover it tends to place within the category of faddists and extremists those who diligently attempt to discover hidden foci and it attempts to vaunt the indolent skeptic who is unwilling to seek the truth.

In the New York Medical Journal of January 18th, 1919, there appears an article on "The Responsibilities of the Dental and Medical Professions," by O. T. Osborne, Professor of Therapeutics in Yale University. This article was refused publication by The Journal of the National Dental Association because it did not pass their Board of Censors.

I do not know how representative of the dental profession this National Journal may be, but the reading of the article in a Medical Journal, with the footnote stating it had been refused publication in a dental journal, is not calculated to bring about the cordial spirit of cooperation which is to be desired.

Osborne states, and I believe truly, that "The platform on which every physician and dentist must stand, no matter what the disease may be, and no matter what the cause of the disease, and no matter how neglible the part that infected teeth and gums play in the disease or disturbed condition, it is dangerous and a menace to the patient and to his family, to allow him to be a carrier of germs of infection. Hence it is the physician's duty to order the surgical removal of mouth infection and it is the dentist's duty to make an infected mouth clean."

Osborne further states: "We are now suffering from the wonderful preservative of modern dentistry, but we have passed the age when indiscriminate killing of sound teeth for crowns and birdgework is excusable. I leave it for the dental profession to decide the relative danger of devitalizing teeth and properly filling the canals, or of leaving the teeth alive for crowns and bridgework. Some members of the dental profession frankly state that to devitalize a tooth except in rare instances is a crime and others state that the abutment teeth of bridges can only last a few years at most on account of the strain they must endure."

It would be quite an ideal condition if the physician could simply refer his patient to the patient's own dentist with the assurance that all possible foci of infection would be recognized and removed. It has truly been observed, however, that, "after the usual attention by the average dental practitioner there still remains in and about the teeth infection sufficient to induce a focal area with eventful serious consequences to the entire body." This leaves the physician face to face with the question of directing his patient to someone whom he regards as a specialist in this branch of dentistry and I believe the proper solution of that phase of the question is squarely to meet it by sending his patient to one who will view the matter purely from a surgical rather than a cosmetic point of view.

The fact that prompt recovery of all ailments does not immediately follow removal of infected teeth does not prove the fallacy of the procedure. Very often there are secondary foci remote from the tooth and sometimes the work is incompletely done. So often have I witnessed ill effects following old fashioned methods of extraction that I have a very warm spot for any technic which aims at modern surgical standards.

Dentists and physicians are constantly drawn closer and closer in consultation over life and death problems. It is most important that a common understanding be reached as promptly as possible concerning the safety of retaining dead teeth. But before and above all let us urge a common meeting upon the platform that all septic conditions of the mouth be made a subject of routine inquiry by the physician and a matter of thoro surgical treatment by the dentist.

Professor Karl F. Meyer.

I have been called on to speak without any preparation whatever, but I will attempt to discuss some phases of focal infection in their application to this symposium.

You probably know the whole question of focal infection was initiated thru the work of Rosenow and Billings. The results of animal inoculations made with streptococcus strains isolated from human focal lesions proved in the mind of these workers the existence of specifically elective types of bacteria. In the light of more recent investigations it is impossible to accept the far-reaching conclusions made by Rosenow and his pupils. The analysis of the experimental data clearly shows that certain definite factors had to be fulfilled, namely large dosage of bacteria, mixtures of streptococci with other bacteria and recently isolated strains. Adhering to these principles it is not difficult to produce focal lesions in animals. The question only arises: Are the inoculated streptococci or the organs of the experimental animals selective in the formation of lesions? Based on my own studies I suspect the selective action of the organs.

I have experimented with streptococci to a limited degree, but with the organisms of the typhoid, para-typhoid and the colon group, and I have been surprised to find focal lesions identical with those produced by streptococci. It is, in my experience, the tissues and the organs of the animal which produce the focal lesions, and not the micro-organisms. I have been particularly interested in the reproduction of cholecystitis, with the aim to treat these acute or chronic infections chemotherapeutically.

It is difficult to produce a cholecystitis of a sufficiently long duration in the rabbit and cat by direct intravenous infection. I therefore planned a reproduction of the conditions as they are found in man, namely, a locus of least resistance, by placing aseptically a small piece of a cholesterinstone in the gall bladder of several rabbits. I left the animals alone for three or four weeks until recovered from the operation. Then I inoculated them with typhoid, or para-typhoid organisms as the case may be. In some instances the dose chosen for the infection was too large and some of the animals succumbed to the intoxication in the next 16 to 24 hours subsequent to the inoculation. Autopsies showed a beautiful cholecystitis, which on cultural tests was caused by streptococci. I then introduced more gall stones, urinary stones, etc., into the gall bladder of another series of rabbits and sacrificed the animals after intervals of from one to three months, and in every instance streptococci were found in the gall bladder wall, even in some cases in the bile. Why? The streptococcus, is, as I have repeatedly expressed, an organism of opportunity and not of specificity. Biochemical and serological tests proved that the streptococci found in the experimental gall bladder infections originated from the gastro-intestinal tract. They

wandered in there by way of the lymphatics or by way of the common duct into a predisposed, mechanically impaired gall bladder. The environment caused by a stone and a silk ligature was most favorable for "secondary invasion."

The next point raised by the papers presented this evening is: Are the streptococci in apical abscesses as potential a menace to health as we are told they are? Do you think the human body has so poor a resistance and such a weak mechanism of defense that it could not take care of these autochthonic streptococci and keep them in check? This is inconceivable as far as experience has taught us. There is always response to these infections. It probably is an immunity of equilibrium. As long as the microorganisms are there and develop, as in malaria or other chronic infections, there is generated a state of immunity. On account of another infection or a physiologic disturbance in the body, the immune substances produced against the streptococci in the focus are spent, and an active proliferation with all its clinical sequelae will be in evidence. As an example, I remember certain features in the buccal cavity of one of my Swiss mountain guides many years ago. He was a giant in his ability to climb the rocks and in cutting steps in glacial ice. He was once guiding me and cut 360 steps in 2½ hours as tho it were a joke! (I was nearly dead after cutting fifteen.) This fellow had two or three apical abscesses, or gum boils as he designated them. They never discharged as long as he was in the mountains, in the prime of health, and his immunity to the bacteria in these abscesses was perfect. At home in the winter, sitting by the fire, he discharged pus profusely—he had apparently no defensive mechanism whatsoever. How far we can apply this observation to the problem this evening is to be discussed some day at length, but I want to recall to you another feature which is generally overlooked. The buccal cavity, on account of its most remarkable power of defense is immune to an enormous array of bacteria. Irrespective of depth or character of the injury, wounds, etc., heal remarkably well in the buccal cavity. Take the organisms which grow in the mouth and place them in the peritoneal cavity or renal pelvis and study the result; the latter cavities have no resistance to these organisms and are rapidly invaded and destroyed by same bacteria which are harmless to the buccal cavity. We have here an example of certain immune forces we do not understand as yet. Our knowledge concerning this local cellular or tissue immunity is incomplete. We talk of leucocytosis, phagocytosis, etc., but we have failed to unravel the mysteries.

A few statements made this evening deserve some consideration. The terms "staphylococci" and "streptococci" have been used extensively and you have been told that certain X-ray shadows are the results of one of the groups of cocci. I would suggest considerable caution in accepting these statements until the laboratory workers have really developed a method by which they can procure specimens from the buccal cavity without the constant fear that they are secondarily contaminated by the omnipresent salivary organisms. It is exceedingly difficult to sterilize the gums or teeth surfaces by the procedures thus far recommended and accurate, convincing tests proving the dependability of the method employed in the culturing of apical abscesses or granulomatas are not available. I always had the impression that many of the so-called apical abscess cultures are the result of a careless technic.

And again the biochemical activities of the cocci in the test tube, for example, proteolysis of gelatine of the staphylococcus, used this evening to explain certain pathogenic lesions around apical abscesses, cannot be considered identical

with the processes occurring in the body. Test tube experiments are frequently artefacts; animal experiments alone will offer the true explanation.

Furthermore, it has been said that we do not know of any chemical which will sterilize infected dental structures without injury to the tissues. Dakin, Carrell and their pupils would probably accept this challenge and refute it clearly by demonstrating that the chlorines and many dyes promptly sterilize tissues without the least injury to the cells. Constantly new chemicals are evolved which specifically destroy bacteria with the least organotropism. Improper composition, reaction and mode of application frequently deprive excellent antiseptics of their value. Hasty criticism as a rule is the result of such mistakes.

I would like to take up many other questions if time allowed, but I hope these few suggestions may perhaps help you to reach an understanding this evening.

Robert Burns, Jr., D. D. S.

Mr. Chairman, Ladies and Gentlemen: I have been granted fifteen minutes to speak on a subject which I would like fifteen hours to cover!

There is no question that a focus of infection is a dangerous thing, whether in toe, tonsil or tooth—or any place else. I will quote from an address by Sir Wm. Hunter, Physician and Lecturer on Pathology to The Charing Cross Hospital, London England, delivered at the opening of the session of the Faculty of Medicine of McGill University. That article was originally published in the London Lancet Jan. 14, 1911. Subsequently it was published in the Dental Cosmos and I would most earnestly commend it to your attention. I particularly commend it to the attention of the gentlemen who take this subject of tooth infection rather lightly and frivolously and make of it a subject of levity.

My clinical experience satisfies me that if oral spesis (and naso-pharyngeal) could be successfully excluded, the other channels by which "medical sepsis" gains entrance into the body might be almost ignored, I speak from experience. The worst cases of anemia, gastritis, colitis of all kinds and degrees, of obscure fever of unknown origin, of purpura, of nervous disturbances of all kinds, ranging from mental depression up to actual lesions of the chord, of chronic rheumatic affections, of kidney disease, are those which owe their origin to, or are gravely complicated by the oral sepsis produced in private patients by these gold traps of sepsis. * * * * * The chief feature of this particular oral sepsis is that the whole of it is swallowed, or absorbed into the lymphatics and blood. Unlike the sepsis of open wounds on the outside of the body, none of it is got rid of by free discharge on the surface. The effects of it, therefore, fall upon the whole of the alimentary tract from the tonsils downward. These effects include every degree of tonsilitis and pharyngitis of gastric trouble from functional dyspepsia up to gastritis and gastric ulcer, and every degree and variety of enteritis and colitis, and troubles in adjacent parts—appendicitis. The effects fall in the second place upon the glands (adenitis) on the blood (septic anemia), purpura, fever septicemia, on the joints (arthritis), on the kidneys (nephritis) and on the nervous system. * * * * The most intense anemia, blood fever, hectic fever, and even ulcerative endocarditis may in my experience be produced by even one septic amalgam filling.

Now I want to draw on the board, as well as my artistic ability will permit, the conditions we have in the normal tooth. Then I will endeavor to show the condition when we have removed a pulp, and I am one of the men who say that the removal of the pulp is one of the most dangerous procedures known to dentistry or medicine. Let that represent the enamel (demonstrating at board). We are not much concerned

with the enamel. Over here run the gingiva. Down along here the pericementum, and don't let yourself imagine, as I once heard a medical man say, that the pericementum is the same as the periosteum! It may be "analgous" to a degree, but it is not by any means the same thing as the periosteum. This will represent in brief outline, the pulp chamber, and down here the pulp canal, in which is found what we so often call the "nerve". Do not forget for an instant that that is alone the "nerve." It is the sole blood supply to the greatest part of that tooth, and no histologist of repute will contradict it.. Noyes, Hopewell Smith, Arthur Black—all say that. The erroneously termed nerve is in reality a complete arterial and venous system. Noyes is almost sure there is also a lymphatic system. Along here (indicating) with the inner layer of the cementum, is the layer known as the granular layer of Tomes. It is a mighty important layer! It separates the dentin from the cementum. That is why when the pulp is removed the dentin must die. There is no possibility of blood supply from the pericementum.

This pulp substance is one of the most peculiar substances in the entire body. It runs thru the tooth and passes out at the apex, and someone of the well known men in the profession, I think it was the late Doctor John Callahan, found in one tooth seventy separate and distinct foramina—minute foramina, running out thru the tooth in this way! You think you fill one of them occasionally, don't you? From this pulp chamber radiate to this line here,—the Granular Layer, the dental tubuli, surrounded by the Fibres of Tomes—millions of them! They are hollow, and contain protoplasmic projections of the odontoblastic cells. That furnishes a blood supply to the dentin. Forget about the nerve supply. All of this bulk of dentin is about 28% organic matter. It is as much subject to decay when the blood supply is cut off, as the end of your toe or anything else, and what happens to the tooth when you remove the pulp? It does die, absolutely and positively, because you remove (if you remove it all, so much the worse) the only blood supply that can get into any of this portion here. Listen to Noyes, one of our best dentinal histologists, and agreeing with him, Hopewell Smith, Black and others. Particularly let this point be impressed on the medical men and surgeons.

"The vital function (of the pulp) is the formation of dentin and is performed by the layer of odontoblasts. These cells also, by means of their dentinal fibrils, maintain the same relation to the dentin matrix, that the bone and the cement corpuscles bear to the matrix of bone and cementum. When the pulp is removed from a tooth its dentin becomes dead dentin in the same sense that bone in which the bone corpuscles have been killed is necrosed bone."

That is what you have when you take out a pulp. You get necrosed dentine—analogous to necrosed bone. How many of you would like to have necrosed bone left in your tissues? The only reason that a dead tooth remains passably comfortable for awhile, or partially useful is because not all of that dead dentin—or necrosed bone, is in physiological relation with the rest of the body. In the most favorable cases it may be that only thru the minute apical foramen—or foramina—is there connection with the outer tissue. The contact is slight, nevertheless sure and deadly. If any portion of the cementum has been destroyed or infected the contact is greater and likewise the ill effect greater and more rapid.

Dr. John S. Marshall has an article recently published which tends to show there may be an osmotic action thru the enamel. If that is so, the enamel is not the wonderful protection that we have thought it was.

It may be possible in a small percentage of cases when all conditions are

favorable, if cementum has not been injured by arsenic, broaches, sulphuric acid, etc., that it may close over that tiny foramen there. Then what would you get? Something like a rotten pea in a can. So long as the can remains tight it may be safe. But who of the best Medical Diagnosticians can say when a man's resistance may be broken down! Who will say when the first heart or kidney lesion starts? What was the big lesson of the draft? That a big proportion of the men had heart lesions. It is the easiest thing in the world to be mistaken in diagnosis. Physical diagnosis does not show a great deal. Everything may be negative, and you call the patients neurasthenic and pass them on to the other fellow.

The cementum is, in a measure, analogous to the periosteum. Thru the pericementum the cementum is kept alive. The cementum, once it loses its attachment to the pericementum and becomes necrosed, is absolutely and constantly a necrotic substance; it has no Haversian system and no means of exfoliating or healing itself. It remains there absolutely and positively until it is mechanically and surgically removed.

If anything lowers the vitality of the pericementum, the cementum becomes necrotic or carious, and Black says a suppurative detachment of the pericementum is a positive and continuous one, and cannot be repaired. I will admit that Nature is very kind to a lot of us fools; the bigger the fool the kinder she is to us. You can go into the country and a half dozen of you will drink of the water. Five of you escape but the sixth man gets typhoid. Who can say why? Dr. Alverez says he has seen many cases where the teeth were pulled, and there was no result. I can show him many cases where the teeth were removed, and the patients did improve. The manner of extraction may be a factor. Of that I shall speak later. I agree with him you must not extract ruthlessly, promising the patient he will be well as a result, regardless of what other conditions may be present. That is out of the question. If you are a dentist you must have as much good sense as the Medical men, and you cannot expect that a tooth is the only focus of infection that a body can have. There are many sinuses, tubes, ears, and so on that may be foci of infection. My point is that this is a new thought with reference to teeth. Almost anybody can see enlarged tonsils, and I have heard said the only symptom for removal was a pair of tonsils and next to that, an enlarged pair of tonsils. But you cannot look down into a bad tooth! It has been pretty well established that the tonsils, sinuses, and so on, may be foci of infection, and the average medical man is prepared to diagnose those foci of infection. The average medical man is not, nor is the average dentist in a much better position because we have not had enough experience yet for any man to say positively this is good and this bad in all cases. But we must be guided by the sum of experience that has been had, and we must use what bit of judgment we have. If you rule out every other possible source of infection or cause of illness, then if there are some of these doubtful devitalized teeth, those are the ones I say take out. It is far better that you remove one good tooth than that one patient may have died ten years before his time of an endocarditis.

Dr. Alvarez would take out the bad ones and leave those that are doubtful. If you have two tonsils—one bad and one doubtful, will you leave the doubtful one in? In the presence of symptoms you had better err on the side of taking out the dead tooth, even if the radiograph is negative as the radiograph is not a true means of diagnosis to 99 9-10% of the men who look at them. Many of these little, tiny bits of areas looked upon as sclerotic bone, walled off lesions, etc., are more dan-

gerous than the big ones anybody can see, as anybody would know the big ones are bad.

In the matter of the removal of those teeth. I very frankly believe that the mere pulling of the teeth is a very poor procedure where there are areas of infection, or where there are indications that the periapical tissues may be diseased. It is a surgical procedure. I would like to see the surgeon who, if he found a sequestrum would reach down with a thin pair of forceps and pull his patient all around the operating room and then go down and use the curette and say "I have got it, etc." He would open it up wide, wouldn't he? Nobody can say where the infection stops in bone about a tooth any more than the aurist can do so in the case of mastoid troubles. No man can go down the narrow socket of a tooth with any specially devised curette and do intelligent curetting. The chances are he will do more harm than good. There was an instance in point a few weeks ago. I was operating on the wife of a San Francisco physician. She was very pale, nervous and anemic. She had been thoroly looked over, and everything that could be done had been done. I said such teeth are bad and should come out. The radiogram of this one is not particularly definite but it is dead and I will advise its removal. That tooth happened to be particularly close to the antrum. I did not have stereoscopic radiograms, but even that does not show all you think it will. I removed the buccal plate, taking the tooth out with my fingers. It was immediately over the antrum and every time the patient breathed the antral membrane belled out. Over the membrane were three or four spicules of bone which were free. What would have happened had I used a curette? I would have gotten into the antrum and caused more trouble than had I left it alone. It was a three rooted molar. Suppose we had a single rooted tooth. In one case that I know of, a single rooted tooth was pulled out and curetted as any one can do under such conditions. Radiographic examinations made a considerable period later showed a granuloma at the spot of the former curettement. I had the pleasure of removing the buccal plate over the socket demonstrating clearly the granuloma to the satisfaction of the former operator. He does not now hesitate to remove the buccal plate when indicated.

We contend with the question of free drainage. After the usual tooth "pulling" you do not get drainage, and even if you do I do not believe you are going to get rid of the necrotic tissue anyway in every case. Nature may do a great deal, but is it not better to be as sure as science can make us that we do a clean job? What possible harm is there in making a little incision and removing the little outer process of bone and looking in and seeing what you are doing? You will get better repair, more smooth and rapid healing than if you break down the tissue and leave a lot of particles of bone that will work out and your patients will come back and say "you have broken my jaw!"

As to the possibility of sterilizing the dead tooth. The little tubuli running in there vary from one to three microns in diameter, i. e., one twenty-five thousandth of an inch. Anybody who can get dichloramin-T or any other medicament in there and be sure he is in contact with the dying stuff is some "cracker jack," believe me!

Guy S. Millberry, D. D. S.

Briefly the cause of all this trouble is injury plus infection. Surgeons know what injury plus infection means. With dentists injury plus infection is what we believe to be the cause of dental disease. The results are infection of the pulp canal or investing tissues, and the alveolar and peridental abscess.

We may resort to surgical or therapeutic measures in the treatment of these

cases. The surgical procedure may include the removal of the tooth, or the amputation of the apex of the root which is involved, making way for good drainage, or it may mean that classical operation described by Doctor Burns.

The problem of exfoliation of necrosed bone is no corollary, since a pulpless tooth is not a sequestrum of necrosed bone, for it frequently has, in absolute contact with, and vitally attached to it, a certain amount of living tissue, the peridental membrane which surrounds and supports, and in part nourishes that tooth. People in a perfect state of health carry pulpless teeth for an indefinite time, and we believe these teeth must receive some nourishment. They are not really necrosed bone as we ordinarily view it. In cases where we have abscesses we have regeneration and repair as the result of a surgical interference, just as we have regeneration and repair in operations on other kinds of tissue. Usually wherever this regeneration of bone takes place sclerotic bone is formed, and it is more dense than the ordinary bony tissue found before these abscesses have formed.

In the therapeutic treatment we pursue various methods, using sedatives, normal salt solution, or irritants, formalin, or formo-cresol for instance, which may destroy the bacteria and the living cells. What happens to the bacteria and to the destroyed cells, and to the cells which are lessened in their functional activity as a result of trauma or medication? Does not nature offer a means for eliminating these cells before regeneration and repair takes place, and does not nature assist us in the same manner in therapeutic treatment as in surgical treatment? Radiograms made before and after therapeutic procedures lead us to believe that complete bone regeneration has been the result of this method. Other things being favorable we should give the tooth and the patient a chance at least, and if the therapeutic treatment does not result favorably we can resort to extraction or the classic operation already referred to.

The mechanical filling of the root canal has been illustrated on the black board by Doctor Burns. He did not mention that as time goes on the deposition of secondary dentine gradually fills the pulp chamber and the canals and we have a gradual recession of the pulp tissue until the canals are nearly completely filled. There is little or no nourishment supplied to the tooth internally, yet it is a vital tooth. Wherein does this vital tooth with its pulp chamber and canals nearly completely obliterated by secondary dentine and with practically no blood supply differ from a "dead tooth" with a satisfactory root filling placed in the canals?

When it comes to the question of filling these multiple foramina (these seventy foramina) I wonder if the same dentist who counted the seventy in one tooth also counted the millions of tubuli existing in other teeth, as it seems almost a physical impossibility to isolate seventy minute tubuli at the end of the root of any one tooth. If he found seventy in one tooth would he not hope to find seventy in every tooth in the mouth? If we have as a result of the filling operation, gutta-percha forced thru the apical foramina of any tooth we find a foreign body which nature may countenance but which probably will not be encapsulated. If the root canal is filled to within one-half millimeter of the apex we have a little opening at the end of the root canal where it is assumed infection from the blood stream can take place. If we concede that infection can be carried from the tooth, then we must also concede that infection may travel from the colon or other sources, to the tooth as well. So which is the place of secondary infection the colon or the tooth?

These dentinal tubules are from one to three microns in diameter and three bacilli can travel abreast in almost any of those dentinal tubules. If we have a

half millimeter of unfilled canal at the end of the root, and if that canal is one-half millimeter in diameter, then in that cylindrical opening at the end of the root of the tooth we have plenty of room for about 250,000 bacteria. How many root-canals are filled to within one-half millimeter of the end of the root? Now for another side of the story!

What does nature do? The pericementum is continually active thruout life, and we know it. Noyes has shown that by osteoclastic activity we can get an absorption on the side of a root canal even involving the dentine and by cementoblastic activity the cementum will fill in even the absorbed area in the dentin. Is not this a protection on the part of nature?

Noyes has verbally reported a case where the crown of a tooth was decayed away, and of course infected and the entire area of the gingival end of the root covered with healed gum tissue so that the cementum had folded down over the decayed end of the root and absolutely encased the root in cementum. Is that not nature's protection, and is it not possible that nature in mechanically clogging the end of the root canal can deposit cementum there and seal it? In thousands and thousands of cases of unfilled root canals that are no menace to health and life, does not nature do something to help us out of our difficulties? If every tooth pulp infected by caries means an extracted tooth and dentistry is not able to cope with this problem, then we must acknowledge that the conservation of human teeth is no longer the aim of modern dentistry.

We have a condition, i. e., autoimmunization, where the individual develops a certain immunity to the organisms in his own mouth, but in other mouths they may be a source of danger, and that is why the disinfection of our instruments is a prime necessity. We have hyper-cementosis and bone regeneration as a further protection on the part of nature. There is no Haversian system in the maxillary bones; there is no osteo-myelitis in the maxillary bones; there is no marrow there and osteo-myelitis in the maxillae and mandible is a mis-nomer. We find that infection rarely follows the inferior dental canal but may be diffused or may be circumscribed in the form of granuloma.

If a pulpless tooth is dead bone, it seems to me it cannot be classed in terms of a sequestrum for it gets some nourishment from the investing tissues and sequestra do not. There is little opportunity for any infection as in a sequestrum. The statement has been made that the enamel may permit the transmission of infection by osmosis. It has never been definitely proven that such is the case.

Before a case of absorption at the end of a root or before unfilled root canals can be charged with the responsibility of any systemic disorders, the cause of which is not definitely known, it must be proven, first, that the organisms found within the encysted mass or within the resorbed area at the end of the root are the same as are found in the area in which this systemic disorder is located, and, second, that the heart lesion or arthritis is secondary to the tooth infection and not the tooth infection secondary to the heart lesion or arthritis. Until that proof is established we are not certain that in every case the one is the cause and the other the result. When we know that in all the folds of the intestinal canal, in the little infoldings of the mucosa, pus producing organisms are lodged and constantly endeavor to find their way into the circulatory system, and when we know leucocytes are there to ward them off, shall we say that teeth and toe nails are the only causes of all these systemic disturbances?

Not many of us perhaps carry "dead teeth" but some of us are willing to carry them as long as we feel physically fit. Dr. Arthur Black is one of the

men who discussed this problem with me in Chicago in February. He said "I have a dead central incisor and a dead lateral incisor I have carried for thirty years and I hope to carry them as long as I live for I have not been confined in bed for twenty-four consecutive hours in any one day since I was four years old" and that is more than thirty years ago.

The question of focal infection is being studied at Rochester, Minn., where dental interns carry on the dental work. They are guided by medical investigators and there is a definite rumor that a report to the medical profession that all pulpless teeth must be removed will follow. These dental interns have not had the benefit of long years of clinical experience and are quite likely to be influenced by the experimental pathologist. Is the dental profession ready to accept such a course as the last word in root canal work? Laboratory experience alone will not answer this question and neither will clinical experience.

In closing let me refer to Doctor Pollia's statement that radiographic findings are thoroly dependable, and Doctor Burns' statement that any man who accepts radiographic interpretations in the last issue is wrong. You see there is a question in the ranks of the extremists. Radiograms are valuable as diagnostic aids but in no sense final. We must admit that the statement that dead teeth are a prey to bacterial attack has not been proved, for a "dead tooth" protected by healthy peridental membrane and enamel, is as resistant to infection as a vital tooth is. We must not lose sight of the fact that all proof shows that pyogenic organisms find their way into the pulp after the chemical disintegration of the tooth has taken place and not before.

There is an economic question. The patients have something to say about whether all their teeth are to be taken out or not, and if not before, they will come back and say it afterwards. If it requires a classical operation, the dissection of the tooth from its socket for all cases that need extraction, we have a problem which the dental profession of today and for a long time to come will not be able to cope with. There are so many teeth to be extracted it would require the combined dental service of the entire country, at prohibitive prices to do this work alone. Let us choose the middle of the road in this problem and assist nature first by endeavoring to prevent dental disease and second by conserving and restoring to health those very valuable organs entrusted to our care, the human teeth.

Dr. Burns, in Rebuttal.

I want to touch upon one statement of Dr. Millberry relative to the deposition of secondary dentine, i. e., the following up of the root canal with secondary dentine in a dead tooth. I take issue with him. You will find that tooth vital sooner or later. That filling in of dentine there is the reaction of trauma or inflammation and nature walls off that sort of pulp and you will find a certain percentage of it still vital.

Dr. Millberry, responding to Dr. Burns: I hope you understand me. I said the tooth was still vital, but contained practically no blood vessels.

Dr. Burns: Very well then, we agree. If this periosteum remains vital that tooth is safe, but when that is all dead and there is no source of supply to it, it is not safe.

As to the regeneration of cementum, Noyes says it never will regenerate unless it is in physiological contact, and it is not in physiological contact when you use arsenic, trauma, etc. What may be done in one thousand or one million instances does not prove it is safe procedure. Dr. Arthur Blacks (wife) lost a tooth—the entire member. Immediately the tooth was replaced and there was a reattachment so far as anybody can tell, of the arteries, nerves veins and lymph channels running thru there.

That occurred once. It would hardly be a safe procedure to accept as a fact and rely on in general practice.

Dr. Millberry, addressing Dr. Burns: May I correct you again. That was the wife of Dr. Edmund Noyes, and the incident occurred before he married her when she was about seven years old!

Dr. Burns: All right. I accept the correction. We are as one, on the fundamentals. As to the economic aspect of this matter. This whole question is a very serious one and we must be guided by conditions. But I do not believe any surgeon or dentist would say because Johnnie Smith could not pay to have his appendix removed we will lay him across the table and wash it out with a hose and spit in it for good measure. We have to do the best we can under the circumstances.

Dr. Pollia: I have profound respect for a genius like Dr. Meyer, and far be it for a mite like me to take issue with a bacteriological giant like he is. The statement that the proteolytic power of the staphylococcus is a test tube reaction is *bacteriologically* true; but the result of a staphylococcic infection as any surgeon present can testify, is necrosis and liquefaction caused by the liberation of ferments present in the phagocytes "which have partaken of too hearty a meal" and disintegrated. Clinically this necrosis and liquefaction is none the less a PROTEOLYSIS. As for the cultures, the method of inoculation was according to the best technic, with some modifications. Whenever more than three bacterial families were found, I discarded those cultures as contaminated. I went into this work to disprove some things and I was not anxious to deceive myself in any way.

I cannot agree with Dr. Meyer that Dakin's Solution and the Chloramine group are the perfect antiseptics. In the first place, because I have worked with Dakin's Solution and the Dichloramine-T with a prominent surgeon in this town, and in many cases there wasn't vaseline enough to keep the skin from being irritated, and the skin is the toughest tissue in the body. Which shows how difficult it is to secure an accurate standardization and hold it. Secondly, it is still doubtful whether the action of these substances is due to antiseptic, germicidal or any other property. Most authorities agree that the action is due to the digestion and solution of necrotic material tissue, debris, etc., thus producing an unfavorable soil for bacterial propagation.

As for Dr. Millberry, "Anytime you have the time, I shall be glad to show you those wonderful changes from the beginning perialveolar thickening to the enormous 'abscess' area found in the radiogram."

Dr. Albert F. Lucchetti, President San Francisco District Dental Society: The question of Fletcherizing is of profound interest, some maintaining that Fletcher has been very instrumental in prolonging life and others hold to the opinion that he taught us how to conserve health by teaching a sane way of eating, so I trust that this symposium will at least show us how to Fletcherize our thoughts and opinions, enabling us thereby to believe that the other fellow is not entirely wrong nor ourselves entirely right.

In the preamble of the by-laws of our society we have some sentences reading after this fashion:

"The object of the San Francisco District Dental Society is to unite in a compact organization all eligible dentists, furthermore, to promote dental education and dental science, in addition to making us more useful to humanity and also to try to bring into more harmonious relations our own with allied professions namely, medical, national, state or county," and so we cherish the ardent hope that a symposium of this kind will help in these directions, and that it will have a tendency to bring us a little closer together.

SOME METALLURGICAL ASPECTS OF AMALGAMS.

By Guy S. Millberry, D. D. S., San Francisco, Cal.

(Read before the San Francisco Dental Society.)

IN TOUCHING briefly on the history of amalgams, I do so because it is inseparably connected with the work of Dr. G. V. Black. The value of his work has been questioned by other investigators recently and a new understanding of amalgams has been presented to us.

In all scientific investigation by professional men the general tendency is to disclose all the findings to the profession-at-large. In all scientific investigation by commercial organizations the general tendency is to withhold such information as may give a decisive or equal advantage to a competitive concern.

When G. V. Black's work first appeared in print many manufacturers of dental amalgam alloys sent representatives to him for a course of instruction in amalgam experimentation and the standards he promulgated at that time have been commercially used almost to the present day.

In this day of rapid strides in scientific progress it is only natural and logical that orders of procedure should change, that improvements in methods of experimentation should be made and that the work of yesterday may be proven to be erratic if not wholly in error today. The Hoe Printing Press, which provides us with our daily paper is not the printing press invented by Richard Hoe in 1846.

Today the experimental work of Dr. Black is being questioned and some of his results disproven, not with the idea of discrediting him, but with the view of improving amalgams. It is unfortunate that in some instances a tinge of harsh criticism has been introduced into our literature, because Black's work has an inspiration to us all.

In any of our problems in dentistry it should be our policy to take up the work where our predecessors stopped, and in the light of modern scientific effort correct the misunderstandings of yesterday and evolve a new understanding for tomorrow.

Metallurgical development in the last five years has received a wonderful impetus from the war. For a decade prior to that time metallurgists were carrying on their investigations in the light of a new conception of chemical reactions, viz: the development in the field of physical chemistry.

With this metallurgical development there has come a better understanding of the physical and chemical changes which take place in alloys. Perhaps in no phase of industrial activity has it developed to such a high degree as in the steel industry, where alloys of steel have been produced which have revolutionized the steel industry itself.

There is no industry which makes greater use of amalgam alloys, and none in which a better understanding of their physical and chemical properties should obtain than in dentistry. In gold and silver mining, mercury is used in the

recovery of the previous metals, then its function ceases.

Electrical amalgams of zinc, tin and mercury are used for battery plates and fuses. Mercury was formerly used for silvering mirrors. These are the chief uses of amalgams, outside of dentistry.

It is not surprising that little scientific work has been done by dentists because dentists are not trained to do industrial research in our dental schools. Dentists who have carried on independent work have been trained in other colleges either before or after they pursued their dental studies or they possessed a keen analytical thinking mind, and a maintaining interest in research problems. I have been looking for some work on amalgams from the pen of Dr. Marcus L. Ward, of Ann Arbor, but judge that administrative duties have interfered with his application to the subject. We may soon expect a book from him which is certain to be constructive in character.

The most important investigations on amalgams of recent date have been made by Arthur W. Gray at Caulk's Laboratories, Milford, Delaware, and by the Bureau of Standards, Washington, D. C., Gray's experimental work[1] dealt chiefly with the crushing strength and dimensional changes of amalgam, the latter being classed as reaction expansions, and to quote Gray, he "uses the term in the algebraic sense to include both expansion and contraction."

As I interpret his work, the values which are helpful to us expressed in terms of a common understanding are (1) that practically all previous tests on the resistance to crushing stresses have been inaccurate because of the small dimensions of the cubes tested, the type of apparatus, used and the absence of temperature regulation and control.

He used a 9000 Kg. Olsen Testing Machine, electrically operated and fitted with thermo elements to produce definite temperature gradients. This made it possible to obtain more accurate results than have ever been presented to us before. Amalgam cylinders 10mm. in diameter and 10 mm. in height packed by a mechanical device capable of expressing the mercury out at a pressure ranging from 100 Kg. to 1600 Kg. per circular centimeter were used for the tests.

One might reasonably raise the question that such methods of expressing mercury and the cylinders of such size do not conform to our customary operative procedure, but we must remember that experiments are designed to give comparative results, using a technic which will not permit of grave experimental error.

(2) The time of packing was found to give the best results when limited to two minutes, the variables in results being attributed to temperature and the condensation of moisture on the filings during amalgamation. *More than 4000 tests proved that strength is directly connected with packing pressure.*

(3) Time of trituration, Mercury—alloy ratio, and the age of the amalgam tested were given full consideration. Prolonged trituration showed that more mercury was retained by the mass, but that this excess might be removed by a heavier packing pressure. Shorter trituration time prevents the granules of the alloy from being thoroly impregnated with mercury—excessive amalgamation seems to have no particularly bad effect upon the results provided packing pressure is maintained sufficiently well to express out the surplus. This probably bears out Harper's Theories of a sloppy mix.

(4) Crushing strength decreases with the temperature increase—A definite mix giving way at 5300 Kg. per Cir. cm. at 25°C.—4050 Kg. per Cir. cm. at 45°C. and 2550 Kg. per Cir. cm. at 65°C.—At the 37.5°C., the amalgam tested (I judge 20th Century

[1] Trans. Am. Inst. Min. Eng. Dec. 1918.

Alloy) give an equivalent crushing strength of 65000 per sq. in. At about 70°C. a remarkable change "Evidently due to absorption of heat" says Gray is shown in amalgams at which a markedly decreasing strength change seems to take place.

The strength of an amalgam varies with its silver content, and the time during which a good alloy may stand on a dealer's shelves was shown to have no ill effects upon the stability of an alloy, like results having been obtained in testing alloys a few days old and sixteen months old.

Dimensional changes.

As you know amalgams may expand or contract. Usually they do both and these changes in structure may continue for a long period of time after mixing.

These changes are due primarily to the changes which may occur in any solid solution in which time and temperature are factors. You are quite conversant with the rapidity of diffusion of gases in gases (Expt. 1) and of the diffusion of liquids in liquids (Expt. 2).

You are also familiar with the diffusion of gases in solids as in CO_2 in charcoal or Hydrogen in Palladium. In both instances the gas is dissolved in the solid and is not in chemical combination with it.

The diffusion of liquids in solids is clearly understood in the absorption by most of the salts of water.

Plaster of Paris in setting dissolves water in the formation of a hydrated salt.

The diffusion of a solid thru a solid is just the same except that it takes place more slowly and the surface tension of both solvent and solute is lessened. The surface tension is clearly illustrated in a globule of mercury which refuses to give up its globular form. This of course is most pronounced in liquids.

The diffusion of solids thru solids, called aphantylosis is interestingly reported by Roberts Austen who states that discs of gold and lead were clamped and allowed to stand at a temperature of 18 degrees C for four years. At the end of that time they were found to have adhered and on analysis it was found that the gold had penetrated the lead to the depth of 8 mm.

Gray, while giving Black credit for his work in so far as careful procedure is concerned, points out that the Black micrometer and particularly the Wedelstaedt tube are inaccurate. In the instance of the tube the only opportunity for measuring the physical changes other than those due caused by the expansion or contraction of the tube itself is the upward change. Gray states that "The only reliable procedure for accurate measurements of expansion whether thermal or reaction—is to employ a method which leaves the specimen perfectly free to change its dimensions in the direction of the displacements under examination," and that implies changes in all direction.

Realizing the inaccuracy of the equipment on the market, and by the way G. V. Black after having failed to obtain instruments from expert mechanics for his experimental work was forced to construct them himself, Gray designed a dilatometer, capable of measuring displacements of 1/500,000 of an inch or 0.05 microns, and fitted with a thermostated oil bath for regulating and maintaining a temperature varying from -50 degrees C to 300 degrees C. A telescopic device is used for reading the changes recorded and these readings can be obtained within one minute after the cylinder has been placed in the instrument and can be followed every minute thereafter thruout the day.

Gray has also proven that the dimensional changes in an amalgam filling can be altered at will, that is variable results can be secured by changing the mercury alloy ratio, the time of trituration or modifying the packing pressure.

I believe we are willing to concur in these statements from our own clinical experience.

This controverts Black's statements that shrinkage and expansion is controlled wholly by the proportions and relations of the constituents used and mixed. In most of the tests by Gray there was an initial contraction then a gradual expansion, then a much slower contraction. When the packing pressure was increased the initial contraction and expansion were accelerated but the final results remained about the same.

Increasing the trituration time beyond a certain maximum (about six minutes) produced an increased contraction while lowering the temperature retarded the reaction expansion changes just as it does in all chemical reactions. The mercury alloy ratio changes showed variable results, the packing pressure changes producing the least variations. Annealing the alloys and the size of the alloy particles were given consideration.

Gray summarizes his results in part as follows:

Comparisons of different alloys are worthless unless the experiments are uniformly made and with dependable apparatus.

A fine grained alloy with plenty of mercury fully amalgamated makes a stronger filling than does a less plastic mix, and if it expands slightly during hardening it will make tight fillings.

Any procedure which hastens complete amalgamation and thus accelerates the reaction results necessary to reach a final stable condition in the finished filling is desirable.

A high silver amalgam is more durable in a few hours than a low silver amalgam ever is and finally is 75 per cent stronger than the low. It requires more mercury, which is cheaper, with the high silver alloy and hence makes a more economical filling material.

Difficulty in amalgamation is often due to surface impurities on the alloy particles which can be treated and corrected. A rapid setting amalgam need not have the mercury expressed until just before placing it in the cavity, and if it has begun to harden, it may be softened with a little mercury without loss of strength to the alloy.

The experimental work at the Bureau of Standards[2] was done at the request of the Surgeon General of the Army. It included compression or crushing tests, flow, relative blackening (a possible test for impurities) and setting changes. A description of the procedure with a tabulation of the results show 7 alloys gave a final expansion over the original contraction. Seven gave a final contraction with the exception of two out of twenty-one mixes.

The crushing strength varied from 30,000 lbs. to 51,550 lbs, per sq. in. and the flow was invariable, beginning two hours after amalgamation and continuing for twenty-four hours with a range of from 3.1 to 47.1 per cent.

I wrote to the Surgeon General to determine which alloys are now used and which were discarded hoping to correlate the information by inference with the products on the market and in reply thereto received the following information: "Crandall's Alloy, S. S. White's Truedent Alloy, and Caulk's Twentieth Century Alloy, are authorized for use in the Army by the Army Dental Corps. These alloys were in use in 1918 and none have been rejected since that time."

One singular thing is noted in the above report. There was no significant difference in the electro motive force between amalgams having a variable zinc content ranging from 0% to 5%. This throws a new light on Crandall's[3] contentions in part at least.

Crandall's amalgam alloy is reported

[2] Com. No. B. 711. Aug. 1, 1919.

[3] Standardizing the Amalgam Filling, W. G. Crandall, D. D. S.

to be free from zinc, because of its electrical potential and because the marked change of bulk it imparts to its alloys and its tendency to oxidize, thus modifying the *balance* of the alloy. The low volatilizing of zinc 918 degrees C is given as one of the reasons Silver fusing at 961.5°C. and tin fusing at 232°C. may easily produce an alloy which fuses lower than 918 degrees C, even tho the silver content may be as high as 68%. The brass industry is able to cope with the problem even tho copper fuses are higher than silver. Balanced alloys have been questioned by Gray as being invariably dependable.

McCouley's statements in 1911 are frequently quoted, but I have long left that his experiments could not be fully confirmed in the light of modern scientific achievement.

The experiments by Crandall showing marked depreciation in crushing strength of Zinc Alloys at 150 degrees F. (about 67 degrees C.) at which temperature hot food and drink may be taken into the mouth is very well explained by Gray in the sudden change in amalgams tested at 70 degrees (158 degrees F.) which he attributes to absorption of heat. If this is a serious matter it would necessitate our eliminating hot foods from our diet or amalgam as a filling material.

The statement that relative electro potential differences of alloys with or without zinc exist, is in part borne out by experiment (Expt. 3) and it is a well known fact that some alloys are more readily attacked by a solvent than pure metals, which fact is attributed to the difference in electrical potential, yet the Bureau of Standards experiments throw a somewhat different light on this problem.

The experiments by Crandall made in the mouth where the respective poles in an electric circuit were connected with a gold crown and an amalgam filling and the leads attached to a galvanometer permit of a possible error. About ten years ago I started a series of experiments to determine the potential differences of different filling materials with the thought in mind of completing them in the mouth. I was confronted with the fact that the body is a good conductor so the problem of short circuits and stray currents made the success of such experiments doubtful. There is room for further study here.

As to the composition of alloys we are convinced that high silver alloys with tin and copper are essential, leaving the metal zinc temporily in doubt.

I am convinced after discussing this matter with Dr. Hinkson, who makes all the Truedentalloy for S. S. White Co. and with Mr. Grier, one of the firm of L. D. Caulk & Co. that small portions of zinc are beneficial. These metals with mercury give us five elements to deal with.

What about the internal structure of these amalgams? Solid solutions have been touched upon. Within such solid solutions in amalgams it is possible to have chemical compounds consisting of binary, ternary, quaternary and quinary groups in which Ag. Sn. Cu. Zn. and Hg. may participate in definite percentage to produce chemical compounds.

In any alloy, the lowest freezing mixture is known as a eutectic and it is a chemical compound.

We may thus have an eutectic for 20th century alloy that differs from Trudent alloy and we may have variable eutetics in the same amalgam where several metals are involved.

When a fused alloy as a plastic amalgam has apparently solidified it is possible that the eutectic is still in solution tho this is not permanent in an alloy solidifying at a temperature well above normal. It might perhaps explain the changes in amalgams at 70 degrees C and thus be responsible for the condition known as flow.

Peculiarly in a study of these eutetics,

amalgams have been used because of their low melting point and facility of handling.

Mathiessen and others have proven that alloys containing solid solutions possess a different electrical conductivity than those which do not. Practically all our amalgams represent a mixture containing solid solutions and eutectics.

The experimental work necessary to determine which metals and in what proportions are the most dependable for amalgam operations in the hands of dentists each of whom may pursue a different method of procedure will rest for the present at least with the manufacturers whose ability to hold the business of the dental profession depends largely upon their willingness to standardize and prove their products just as it does in automobiles or shoes.

On the other hand operative procedure on our part should be developed to such a degree that the combined results of skillful effort and dependable material will benefit humanity.

In closing I wish to disclaim any original work in the presentation of this paper. I have drawn freely from such literature as I have been able to secure and present it merely as an interpretation of these data. If it has proven helpful to you, my effort has been fully repaid.

WHY IS THE FIELD OF PROPHYLAXIS MARKED WITH A LACK OF ENTHUSIASM?

By Dorothea A. Howes, D.D.S., Washington, D. C.

(Read before the National Dental Association at Its Twenty-third Annual Session, New Orleans, La., October 20-24, 1919.)

WITHIN a short period of time the dental profession has attained great popularity. Outside of professional life one can't escape the ever present topic of dentistry. Modern literature is full of its importance and bearing upon health. Topics relating to the part dentistry has played in the recent increase in length of life are common in current magazines and newspapers frequently flare the headline, "Death from a Tooth." The pulpit has even taken inspiration in the wonders of orthodontia, weaving sermons of our profession's ability to mould distorted features into lines of strength and normality. At social functions one of the favored topics of conversation is dental troubles and miraculous cures obtained thru scientific dental diagnosis where medical means failed.

It is obvious that a favorable percentage of people have come to appreciate the value of restored lost tooth structures, in the form of fillings, crowns, bridges, etc., and in establishing occlusion so that mastication may be more thoroly performed. In contrast with the realization of the importance of reparation, is the woeful ignorance that exists amongst the vast majority of people toward mouth hygiene. Those who realize the

necessity of reconstructing lost tooth tissue, and insist upon its completion to its every detail that they may retain their dental organs as long as possible, for both health and aesthetic reasons, are passivley content to possess mouths that present a slovenly and inflamed condition with marked signs of gum infection from deposits and fermentative reactions. That this condition may be one eminent cause of the ever occurring decay, malnutrition,—not to mention many chronic and acute diseases,—does not occur to the average person. They brush their teeth twice daily, very often less, make periodical visits to the dentist once each year or twice each year, but with all, the gradual loss of the teeth seem certain and one is most fortunate who by the fortieth year escapes the ravages of pyorrhea and its detrimental results.

Perhaps one of the greatest factors that has helped to make the American people alive to the importance of dentistry in recent years has been the X-ray. This has been of very great value in the art of diagnosing, throwing light on former obscure conditions that baffled both medical and dental men. It has been the means of associating the two professions more closely by emphasizing the fact that many chronic and acute diseases come from mouth infection. It has been met by the laity with a widespread enthusiasm. One might almost say, abscessed teeth and non-vital teeth were "in vogue," so prevailing are their dangers in the minds of the people. That bodily pain in the form of rheumatism, neuritis, arthritis, valvular heart disease and many other diseases has been caused by anything as simple as a tooth, and actually relieved by its extraction has created an appalling astonishment thruout the country. People have become educated to the use of the X-ray thru the unanimous emphasis on the part of the profession, just as they have become acquainted with other forms of dental progress. They are only too willing to be relieved of any possible doubtful conditions that could be easily clarified by the radiograph. There are a few who from their own initiative have regular X-ray examinations as a protection against apical infection. A prevention "better late than never." These same individuals who are so anxious to avoid apical infection, and often rightly concerned about the state of their tonsils, fearful that no pus germs enter their systems, are wholly unmindful that they, more than likely, have mouths in which a low grade infection, perhaps, exists about the gingival margin, only, mildly toxic, but holding full sway to develop into a more severe form, and who, not unlikely, have an infection, equal in toxic absorption, to both diseased tonsils and apical abscesses. Beyond the age of youth a natural healthy oral cavity with a firm, pink, lustrous appearance to the gum tissue is so rare as to be worthy of comment. The fact that more teeth are lost thru pericemental destruction than from any other cause illustrates clearly how few appreciate or realize the necessity of maintaining a normal gum tissue. A fuller appreciation is shown toward an artistically technical bridge or inlay, which are only too often the burden of teeth whose pericemental support is lost below the gingival third of the root, and having swollen and congested gums that were disregarded by indifferent operators. The results are only too apparent.

I have not infrequently had patients, who after a suggestion as to the suppurative condition of their pericementum, remark that they were aware of its existence; that the condition had been present for quite some time; and presumed, as a dentist had once predicted that "some day pyorrhea would inevitably manifest itself." The prevailing idea seems to be that pyorrhea develops with a most astonishing suddenness! Altho their patients were content to resign themselves submissively to the arrival of the "some

day," when pyorrhea would suddenly inflict itself upon them, they were most particular in the meantime, to have every cavity filled and refilled, that the dread of losing their teeth might be postponed as long as possible.

The public attitude is a reflection on the trend that the dental course has taken. Evidently the magnitude of energy has been centered toward the perfecting of a mechanical and curative art, struggling to restore nature's form to health, usefulness, and aestheticness.

While this is important, it does not need to be *all* essential, monopolizing the entire attention to the exclusion of a service that would do far more in the progress of dentistry and likewise the progress of the public in education and health.

Research workers in the medical profession have brought forth vaccines and serums which are universally used in the prevention of many diseases. The profession is not divided in theory as to the success of this prevention they are wholly and nationally agreed.

While the dental profession has long since recognized that all dental diseases are traceable to acid fermentation and production of excess micro-organisms due to uncleanly mouth conditions, there is still no visible sign of any effort to adopt unanimously a solution that will act as a check and prevention. When it has been so successfully proved by the few who have devoted their time to its study, that the only means of abolishing putrid unhygienic mouth condition is by the application of systematic prophylactic treatments, then, why is the Field of Prophylaxis limited, and I might say, antagonized by a part of the profession?

"Do plaques stay removed?" Inquiries one eminent co-worker, "No they return in full force in six hours and in some people's mouths in two hours, showing that the micro-organisms are always there and rightfully there."

"Continued prophylactic treatments are rank nonsense," proclaims another. Scraping and scratching the enamel and gouging the gums to keep a theoretical plaque removed is not only ridiculous but merciless."

Another, incredible though it may seem, declares, "Too frequent polishing is liable to wear off the enamel, the safest way to produce a thoroughly clean mouth is to chew gum!"

These statements, with the prevailing lack of interest evidenced by the astonishing number of mouths deplorable in accumulation of deposits and debris, but in *top-notch condition with reparative work,* is a good reflection of the lack of appreciation and enthusiasm shown toward prophylaxis by the greater part of the profession.

But is prophylaxis the infallible ideal treatment that is has been pictured to be?

No, I do not believe that caries can be absolutely prevented, no matter how early prophylaxis may be started. But this does not lessen its true value—its power to render the beginning and progress of decay extremely difficult, and above all, its power to almost completely annihilate the causes of gum infection and consequent disease that forms one of the greatest problems confronting the profession.

I am sure the imperviousness toward its good is due mostly to the concentration we all give to our own work, overlooking that which does not have any immediate bearing upon it and which without question can be put off, giving more time to emergencies that must be constantly met. Anti-prophylactic workers are very often accused, perhaps rightfully, of a greater concern for their own materialistic welfare than the welfare of humanity. Fortunately prophylaxis is not so much disapproved as disregarded.

If our ideals of striving for the greatest good of our patients be attained, the treatment which is always best for them must be recognized, and if not performed, then its need emphatically ex-

plained. The public will recognize the importance of oral hygiene, as they have that of reparative work, only thru the combined teaching of the profession, and until that time dentistry can not be directed upward and forward and so reach its highest standard.

DISCUSSION.

Arthur H. Merritt, New York.

It has been said that "Ignorance is the curse of God; knowledge the wings whereby we fly to Heaven." This is probably one of the answers to the question raised in Dr. Howe's paper. Not a little of this ignorance is within the dental profession itself, which explains why it is that so often they neglect the hygiene of their patients' mouths. People do not go about with unclean and unhealthy mouths because they enjoy it, or because they are unwilling to pay for proper care as some seem to think, but simply because they are ignorant in the matter. There is also another answer to Dr. Howe's question: there is nothing spectacular about prevention. The dentist who makes beautiful inlays and elaborate artificial restorations, has made an impression upon his patient that would not have been made had he, by proper attention to oral prophylaxis, prevented the need for these same restorations.

Until we, as a profession, preach in season and out of season, the gospel of clean mouths, and practice what we preach, there is bound to be more or less indifference on the part of our patients, with nobody to blame but ourselves.

Hertha Hartwig.

I think Dr. Howe's paper a fine and and timely, too, because we most certainly do need to have drawn to our attention the lack of enthusiasm and our own very probable responsibility for it.

Some dentists think that if they have a monument along restorative lines in their patients' mouths, they are doing the greatest thing. These monuments are the things which speak for them, while with prophylaxis and all this preventive work, there is need of so much eloquence to prove the benefits of a service which can not be measured in so many square millimeters of gold and porcelain that most dentists feel it too great an effort. Until they awaken to their own value and responsibility as great educational factors, it can not be hoped that they will wax enthusiastic over this work.

None of us realize how much all this means to the patient. Talk prophylaxis, which you must not forget means cooperation at home, to a patient who has never heard it before. It probably hasn't even occurred to him that such careful attention to the mouth is either necessary or so conducive to general well-being, especially in the case of children. He will be amazed and then enthusiastic and in turn, imbue other patients with enthusiasm. Every time they realize what it means and what a great thing it is, they will become the greatest of educational factors themselves. It can be likened to a pebble dropped into the water, with the ensuing ever widening circle. The more people are educated to what the prevention of periodontal disease means and at the same time, prevention of caries, the more eager will they become for knowledge of the physiology of the mouth and teeth, than which nothing is more desirable, because it shifts the greater responsibilty for oral health onto the patients' shoulders.

The time is to come—probably in the millenium— when a crown or a bridge in any patient's mouth will be a disgrace to the patient and to the dentist who put it there.

E. J. Weaver, Milwaukee, Wis.

The lady who has just spoken has put me in mind of an incident. I can hardly keep from briefly stating it.

Before I state that instance, I want to compliment Dr. Howes on her paper.

Papers of this nature are the only ones which should be read before the general dental societies, which will make them stop and think of prophylaxis as being perhaps worth while. They have to be made to think.

It was my fortune or misfortune, to have charge of the prophylactic work during our state dental society, a few months ago. In that unit, we had two man working from the standpoint of the general practitioner. It was a fact, neither of these men had ever practiced prophylaxis to speak of at all, more than perhaps the cleaning of a set of teeth for which they may have received a very small fee. Simply being with our unit and hearing discussions on the way we were laying out the work evidently set them to thinking.

Before we were thru with that work, which went over a number of weeks, first one of the men came to us one day and said, "Do you know, I thought I would try out that scheme on a patient of mine. In place of cleaning his teeth for fifteen minutes, I spent two hours. I had him come twice and to the best of my ability I went over those teeth. When I got thru the man said to me, 'Say, what have you been doing to my mouth?' I said, 'I have been trying a little bit of prophylactic work.' 'For heaven's sake, why didn't you do that before? My mouth feels clean for the first time in twenty years,' he exclaimed. That states about what the patient thinks.

The second man said: "I didn't think it was ever possible to get any money for cleaning teeth." I won't state the amount of money which those fellows got for the prophylaxis, but it was as much or more than their time was usually worth. What the patient told the second man simply repeats the experience of the first.

These men didn't understand how to educate the patient as to the value of what they were attempting to do.

Dr. I. Hirschfield, New York: I just want to compliment the last speaker. They immediately lost confidence in the dentist of the past because he didn't give it to them. I very often have trouble keeping those patients with those dentists because of their loss of confidence in them.

Dr. Howes: I would just like to thank the members for their kind appreciation.

THE INFLUENCE OF THE SURFACE TENSION OF THE CULTURE MEDIUM ON THE GROWTH OF BACTERIA.*

By W. P. Larson, W. F. Cantwell and T. B. Hartzell.

(From the Department of Bacteriology and Immunology, University of Minnesota, Minnesota.)

THERE is probably no phase of bacteriologic research that has occupied the attention of investigators more than the preparation of culture mediums. Most of such investigations have been directed toward supplying bacteria with the nutritive material best adapted to their specific individual requirements. Aside from its reaction, the physico-chemical state of the medium has received but little attention.

It is the purpose of this paper to record the behavior of some of the common bacteria when cultivated on medium of low surface tension.

Our investigation naturally began with a search for suitable substances with which to lower the surface tension of mediums. It was soon found that substances that would lower the surface tension of water would not always act satisfactorily in lowering the tension of broth or other mediums. Furthermore, only such surface tension depressants could be used as were of themselves toxic neither to bacteria nor to laboratory animals into which cultures or filtrates were subsequently to be injected. It was found that soap fulfilled our requirements better than anything else tried. A large variety of commercial soaps were used, after first being purified and neutralized. They all possessed certain disadvantages, however, as they invariably caused a marked turbidity of the medium. Furthermore, the surface tension of the medium depressed by the various soaps would not remain constant, due to the fact that soap tends to hydrolyze when diluted, which causes the tension to rise. Studies were therefore made in an effort to secure a soap that would be perfectly clear when in solution, and which would be sufficiently stable to prevent the objectionable fluctuations in the surface tension of the medium. It was found that pure castor oil saponified with NaOH met these requirements.

A number of neutralized fatty acids form a clear solution when hot, but become turbid and viscous on cooling. Solutions of castor oil soap on the other hand remain perfectly clear when cooled, do not hydrolyze on sterilization in flowing steam, nor do they become viscous when cooled.

Castor oil soap is prepared by adding an alcoholic solution of NaOH to castor oil slightly in excess of its saponification equivalent. The mixture is then heated over the water-bath until saponification is complete when the soap is dissolved in hot absolute alcohol to which is added a few drops of phenolphthalein. Hydrochloric acid, diluted with alcohol, is then added drop by drop until the soap solution is neutral. It is then filtered on paper to remove the chlorids. The excess of alcohol may then be removed by evaporation, after which the soap solution is poured into a saturated solution of NaCl to remove the glycerol and other impurities. After further filtration followed by washing with a saturated solution of sodium chlorid the soap is ready for use.

*Aided by a Grant from the National Dental Research Association.

The drop method was used in measuring the surface tension.

$$\frac{\text{Weight of number drops of medium}}{\text{Weight of number drops of water}} \times 73$$

In order to give a general idea of the comparative surface tension of various fluids it may be stated that that of water is 73, alcohol 22, and ether 16 dynes.

The broth prepared in our laboratory by standard methods has a surface tension of about 59 dynes. This may vary slightly, but it was always found to be between 58 and 60 dynes, 59 being the value usually obtained. The surface tension of broth may be lowered to any desired point down to 32 dynes by adding the necessary quantity of soap solution to the medium.

In growing bacteria on broth of lower surface tension we were impressed by the behavior of the B. subtilis when the tension had been depressed below 45 dynes. When grown on such medium this organism no longer forms a pellicle, but grows down in the body of the medium. When the tension of the broth is depressed to below 45 dynes the growth of the B. subtilis is largely at the bottom of the tube. It was further observed that spore formation was markedly suppressed and sometimes entirely absent, especially after cultivation on low tension mediums for several generations. B. subtilis grown on medium of low surface tension may sometimes be rendered sterile by heating to 60 degrees for an hour which indicates that spore formation has been entirely suppressed. It is generally supposed that B. subtilis grows on the surface of broth because of its great demand for oxygen.

Since our work has shown that this organism is capable of growing thruout the medium, even at the bottom of the tube, it is evident that its oxygen requirement is not the chief reason for surface growth on ordinary broth. In this connection we would not overlook the fact that lowering the surface tension of water increases its ability to dissolve oxygen and other gases. A striking feature common to most bacteria which grow on the surface of liquid mediums, particularly B. subtilis, B. mesentericus, and B. tuberculosis and others, is that whenever there is pellicle formation there is little or no growth except at the very surface. In inoculating a flask of glycerol broth with B. tuberculosis large numbers of organisms may sink to the bottom of the flask, but only those that remain at the surface show signs of multiplying. Other bacteria, such as B. coli, the cholera spirillum, first show signs of development at or near the surface even tho there is no pellicle formation. What can be the explanation of this selective site of so many bacteria? It is possible that the surface of the medium certain substances are concentrated which favor, or are even necessary, for the metabolism of many microorganisms. It is a well known fact that surface energy tends to reduce itself to a minimum, and that this may be accomplished in two ways: (1) by the reduction of the surface area of the liquid, or (2) by reducing the surface tension, or both. The surface tension of the liquid is reduced to the lowest point possible thru the concentration of the surface tension depressants at the surface of the liquid. B. subtilis and other bacteria, which show such predilection for the surface of the medium, probably select this zone because the surface tension reducing substance concentrated there are required for their metabolism. The pellicle formation is probably a mere coincident, the surface tension of the fluid being sufficient to support the weight of the organisms just as certain insects may be supported on the surface of water because of its high surface tension.

The question naturally suggests itself as to whether or not all aerobic bacteria might not become pellicle formers were the surface tension of the culture medium sufficiently high. Since it is impossible to raise the tension of a me-

dium much above that of water, this experiment has its limitations. However, we have found that B. coli as well as some of the paratyphoid bacteria form well developed pellicles when grown on an inorganic medium having a surface tension of 72.2 dynes.

Investigations have also conducted with reference to the influence of the surface tension of the medium on some of the common anaerobic bacteria, such as B. tetani, B. botulinus, B. chauvei. It was found that B. tetani grew well under ordinary aerobic conditions proving the surface tension of the medium had been properly depressed. B. tetani produces a potent toxin when grown on ordinary broth, the surface tension of which has been reduced to about 40 dynes.

This would seem to indicate that we cannot draw a sharp line between the aerobic and anaerobic bacteria altho there can be no question but that some bacteria demand a liberal supply of oxygen, while others cannot thrive where the supply is too liberal. In this connection it may be pointed out that one of the popular methods of producing anaerobiosis is to cover the medium with a layer of sterile oil, the object of the oil being to exclude oxygen from the medium which it covers.

Since oil is a much better solvent of oxygen than water, is it reasonable to suppose that oil actually does exclude oxygen? If the tube were sealed off immediately after introducing oil into the test tube it might be argued that the oil, because of its being a better oxygen solvent than the medium, would cause the gas to diffuse from the medium into the oil later; but since the sealing of the tube following the introduction of the oil is not necessary to effect the so-called anaerobic condition, this is obviously not the explanation. A little experiment which shows that the oil does not exclude oxygen from culture medium may be made by inoculating a tube of broth with a culture of B. subtilis after which the inoculated medium may be covered with a layer of sterile oil; 48 hours following this inoculation it will be found that the subtilis has formed a very heavy pellicle just beneath the oil layer, showing that oxygen does pass from the oil into the medium, otherwise this organism would not be able to grow at this point. The oil undoubtedly favors the growth of anaerobic bacteria because it reduces the surface tension of the medium, and as a result the surface tension depressants contained in the medium are not concentrated at the surface as would ordinarily be the case, but are dispersed thruout the medium where they can serve the metabolic requirements of the anaerobic bacteria that under ordinary circumstances would be able to avail themselves of these surface tension lowering substances because of the excess of oxygen at the surface where they are concentrated.

The mere act of lowering the surface tension of the medium will not cause all anaerobes to grow under ordinary aerobic conditions. Such a germ as B. chauvei fails to show any appreciable development under such conditions.

Most bacteria seem to grow best when the surface tension has not been depressed below 53 to 55 dynes, altho most of the more common bacteria, such as the staphylococcus, B. coli, B. typhosus, and B. paratyphosus show some growth when the surface tension has been depressed as low as 32 dynes.

Surface tension below 45 dynes tends to cause many bacteria to agglutinate, thus giving the impression that the growth is entirely at the bottom of the test tube this being particularly true of the typhoid and paratyphoid bacilli. The streptococci and pneumococci do not grow well where the surface tension has been brought below 50 dynes. The pneumococci show a tendency to disintegrate when grown on a medium of a low surface tension. This observation suggests that the solution of pneumococci by bile may be due, in part at least,

to the lowering of the surface tension which bile induces. B. tuberculosis grows very well on medium of low surface tension. Like B. subtilis it no longer grows with pellicle formation, but grows at the bottom of the medium.

The pathogenicity of bacteria seems to be profoundly affected by the surface tension of the medium on which they are grown. We hope to give a detailed report on the relation of pathogenicity and toxin formation of bacteria to the surface tension of the medium in a subsequent article.

SUMMARY.

The growth of bacteria on ordinary broth is greatly influenced by the surface tension of the medium.

A sodium soap of castor oil proved to be a practical surface tension depressant.

All pellicle formers cease to grow at the surface where the tension is below 45 dynes.

The tendency of B. subtilis to form spores is considerably reduced when the organism is grown on broth medium of low surface tension.

Some anaerobes, B. tetani in particular, grow well under ordinary aerobic conditions on medium of reduced surface tension. The layer of oil often used to cover mediums for the purpose of producing anaerobiosis, probably acts by reducing the surface tension rather than by excluding oxygen.

EFFECT OF FOREIGN PROTEIN ON THE KIDNEY.

By E. T. Bell and T. B. Hartzell.

(From the Department of Pathology, University of Minnesota.)

IT IS known that some persons develop symptoms of anaphylactic character after eating certain foods, of which shellfish, egg-white, and certain fruits are familiar examples. It is believed by many that asthma, urticaria and hay-fever have their origin largely in a condition of hypersensitiveness to special foreign proteins. The symptoms produced when a foreign protein is introduced into a sensitized animal are usually referred to a hypothetical substance—"anaphylatoxin"—formed by the action of antibody on antigen. There are reports in the literature to the effect that anatomic changes in the tissues occur in acute anaphylaxis, and Longcope[1] has advanced the theory that repeated anaphylactic shock may be responsible for such chronic diseases as nephritis. By a series of animal experiments Longcope believes that he has shown that repeated nonfatal poisoning with foreign protein is responsible for some cases of chronic nephritis, cirrhosis of the liver, and chronic myocarditis. Our interest in the etiology of chronic nephritis led us to repeat some of his experiments.

ANATOMIC CHANGES IN ACUTE ANAPHYLAXIS.

Gay and Southard[2] described hemorrhages and areas of fatty degeneration in guinea-pigs that died in acute anaphylactic shock and in those killed subsequent to severe shock. The hemorrhages were widely distributed, but

[1]Jour. Exper. Med., 1913, 18, p. 678; 1915, 22, p. 793; Trans. Assn. Am. Phys., 1914, 29, p. 49.

[2]Jour. Med. Research, 1907, 16, p. 143; 1908, 18, p. 17.

were most commonly found in the mucosa of the stomach and in the lungs. Areas of fatty degeneration were found in capillary endothelium, in the mucosa of the stomach, and in the cardiac and voluntary muscle. The hemorrhages were interpreted as the result of the degeneration of the capillary endothelium. But Anderson and Rosenau[3] in repeating this work could find no evidence of fatty degeneration. They did not find the hemorrhages so constantly and seem inclined to interpret them, when present, as due to the violent struggling of the animal during shock rather than to the action of the protein.

It is known also that fatty degeneration does not occur suddenly. Usually several days are required for a cell to become definitely fatty, and very little fat accumulates earlier than 48 hours after the injury. It is inconceivable that this change could occur within a few minutes or even within a few hours. Again the normal variations in the fat content of voluntary muscle fibers are so great that one can seldom speak with assurance of a fatty degneration in this tissue. These considerations lend strong support to the negative findings of Anderson and Rosenau.

The Arthus[4] phenomenon seems to lend experimental support to the view that foreign protein may produce local anatomic changes when introduced into a sensitive animal.

Longcope[5] studied the effects of repeated injections of foreign protein in guinea-pigs, rabbits, dogs and cats. Small sensitizing doses of horse serum or egg-white were given 20-25 days before the secondary injections were begun. The secondary injections were spaced and regulated with the object of producing shock a number of times. Some of the experiments lasted as long as 3 months, during which time as many as 15 injections were given. Not all injections resulted in anaphylactic symptoms and many of the reactions were recorded as slight.

Changes were found in the kidneys in 23 of 29 rabbits, and in a large proportion of the other animals experimented on. In the animals that died acutely diffuse degenerative changes were found. In those that lived longer, extensive round cell infiltration was the most striking change. Dense fibrous rays with atrophic tubules were found in 6 rabbits. Six rabbits showed normal kidneys at necropsy. Portal cirrhosis of the liver and areas of chronic myocarditis were also found frequently.

In a more recent paper Longcope[6] found that similar tho less marked renal lesions may be produced by a single large injection of foreign protein, or by several injections in which a different protein was used each time. All the sensitized animals developed nephritis, but of those that received a single large injection, or several different proteins, only one-third developed nephritis. He concluded that foreign protein is itself toxic and many produce nephritis, but that previous sensitization increases the extent, severity and constancy of the lesions.

The lesions described by Longcope correspond in all respects with those of ordinary spontaneous nephritis. As controls, 24 supposedly normal rabbits were studied, and in 4 of these a few small areas of round cell infiltration were found similar to those occurring in the experimental animals. The argument that the lesions were produced by the foreign protein because they were more extensive and severe than in the controls has no value, since advanced stages of spontaneous nephritis are of common occurrence. The controls apparently did not have the same opportunity to contract an infection, since there is no mention of their having been kept under

[3]Jour. Med. Research, 1908, 19, p. 57.

[4]Arch. internat. de Physiol., 1908-9, 7, p. 471.

[5]Jour. Exper. Med., 1913, 18, p. 678.

[6]Ibid., 1915, 22, p. 793.

the same conditions and for the same length of time as the experimental animals. It is also to be noted that some of the animals may have had nephritis at the outset of the experiment, since it is admitted that animals with a faint trace of albumin were used. Again, there was no control at all for the healed stages of nephritis, of which 6 cases are mentioned and attributed to the action of the protein, since these do not cause albuminuria. No cultures were taken from the kidneys to determine whether the nephritis was due to infective or to chemical irritants.

Three of Longcope's experiments were controlled somewhat better. Three dogs were operated on, and a portion of one kidney from each was removed for examination before the secondary inoculations were begun. The excised portions of the kidneys were found normal, but each animal developed nephritis after a number of injections of protein subsequent to the operation. But no cultures were made from these kidneys and a renal infection was therefore not excluded.

The uncertainty of the results of Longcope's work is indicated further by his attributing the ordinary cirrhosis of the liver of rabbits to the action of the protein. This is an unusually common lesion. It occurs in fully three-fourths of adult rabbits and is probably due to bacterial infection. All who have studied rabbit livers extensively recognize this as a spontaneous lesion.

Longcope's conclusions were confirmed by Boughton[7] to the extent that repeated injections of foreign protein may produce renal lesions. Boughton produced repeated anaphylactic shock in guinea-pigs, using beef serum and egg-white as antigens. Some of the animals received as many as 19 secondary injections, and the average number of injections was 9. The average duration of the experiments was 4 months, but some animals were under experimentation as long as 8 months. The urine was not examined systematically. Twenty-three guinea-pigs were used. Degeneration, desquamation, and necrosis of the tubular epithelium was the most striking change noted and was seen in all cases. Round cell infiltration was found in nearly all cases, tho it was never very marked. There was no distinct scar formation.

Six controls were given injections of protein so spaced as to avoid the production of anaphylaxis. Tubular injury was much less marked than in the sensitized animals. Areas of round cell infiltration were also seen, but these were only one-tenth to one-fifteenth as frequent as in the sensitized animals.

All injections were given intraperitoneally and ranged as high as 10 c c (undiluted serum, or 50% solution of egg-white). The kidney lesions were regarded as subacute rather than chronic.

Boughton seems to conclude that foreign protein produces some renal injury when injected without the production of anaphylactic shock, and rather severe injury when this phenomenon is repeatedly produced. The tubular injury was the most prominent lesion, and the production of a typical chronic nephritis is apparently not claimed. As regards the interpretation of the round cell infiltration, the sources of error are the same as in Longcope's experiments.

Experiments.

The purpose of our investigation was to determine whether chronic nephritis can be experimentally produced in rabbits by a foreign protein. Repeated shock was produced in a few animals, but in most of the experiments no attempt was made to produce anaphylaxis.

Special effort has been made to eliminate various sources of error. All rabbits that had albuminuria were discarded. In order to eliminate the occasional inactive cases of spontaneous nephritis, rabbits with normal urine

[7] Jour. Immunology, 1916, 1, p. 105.

were operated on with aseptic precaution under ether anesthesia in order to expose and examine the surface of the left kidney. All except early cases of nephritis may be recognized by the presence of spots and pits on the surface of the kidney. Both kidneys are nearly involved to about the same extent, so that it is not necessary to examine the right kidney. About one week after the operation, the experiment was begun in the case the wound was completely healed and the urine free from albumin. Ascitic fluid, egg-albumin, and sheep serum were used. Injections were made intravenously except as otherwise indicated.

ASCITIC FLUID WITH ANAPHYLAXIS.

Rabbit 25.—Weight 2,000 gm. July 3: Test for albumin negative. Left kidney exposed and found normal.

July 12, 17, 24: 1c c sensitizing injections of ascitic fluid.

Sept. 10, 1 c c, moderate anaphylactic symptoms.

Sept. 21: 0.5 c c, slight symptoms.
Oct. 5: 0.25 c c, slight symptoms.
Oct. 15: 0.5 c c, slight symptoms.
Oct. 25: 0.5 c c, moderate symptoms.
Nov. 5: 0.5 c c, moderate symptoms.
Nov. 15: 1 c c, severe symptoms.
Nov. 21: Found dead.

Tests for albumin negative, Sept. 10, 25, Oct. 11. Trace of albumin, Nov. 7. Seven secondary injections were given over a period of 71 days. At necropsy the kidneys showed no gross changes. Microscopically an occasional small focus of lymphocytes was found deep in the cortex.

Rabbit 35.—Weight 1,885 gm. Sept. 11: Test for albumin negative. Left kidney exposed and found normal.

Oct. 5: 0.25 c c, ascitic fluid.
Oct. 15: 0.5 c c, slight symptoms.
Oct. 25: 0.5 c c, moderate symptoms.
Nov. 5: 1 c c, slight symptoms.
Nov. 15 1 c c, slight symptoms.
Nov. 26: 2 c c, slight symptoms.
Dec. 6: 2.5 c c, moderate symptoms.
Dec. 17: 2.5 c c, slight symptoms.
Dec. 27: 2.5 c c, slight symptoms.
Jan. 10: Killed.

Tests for albumin negative, Nov. 8; positive, Jan. 4 and 10. On Dec. 5, the serum from this rabbit gave a strong precipitin test with human blood serum. Eight secondary injections were given over a period of 87 days. At necropsy the kidneys showed no gross changes. Microscopically and occasional small lymphocytic focus was found in the cortex.

Rabbit 33.—Weight 2,320 gm. July 18: Test for albumin negative. Left kidney exposed and found normal.

Sept. 10: 0.5 c c, ascitic fluid.
Sept. 21: 0.5 c c, severe symptoms.
Oct. 5: 0.25 c c, slight symptoms.
Oct. 15: 0.25 c c, slight symptoms.
Oct. 25: 0.5 c c, moderate symptoms.
Nov. 5: 0.5 c c, moderate symptoms.
Nov. 15: 1 c c, slight symptoms.
Nov. 26: 2 c c, slight symptoms.
Dec. 6: 2.5 c c, slight symptoms.
Dec. 17: Found dead.

Test for albumin negative, Sept. 10, 25, Oct. 11, Nov. 7. On Dec. 4 the serum from this rabbit gave no precipitate with human blood serum. Eight secondary injections were given over a period of 87 days. The kidneys showed no lesions grossly or microscopically.

ASCITIC FLUID WITHOUT ANAPHYLAXIS.

Rabbit 32.—Weight 2,320 gm. Oct. 2: Test for albumin negative.

Oct. 6: Left kidney exposed and found normal.

One c c ascitic fluid, Oct. 16, 23.
Two c c, Oct. 27, Nov. 1, 6, 12, 17.
Three c c, Nov. 22.
3.5 c c, Nov. 27, Dec. 3, 8, 13, 18, 22, 26, 31.

Tests for albumin negative, Oct. 13, Nov. 9, 22, Dec. 22, Dec. 13; positive, Jan. 3.

Killed, Jan. 3. 16 injections were given over a period of 79 days.

No gross changes found in the kidneys. Microscopically a few small lymphocytic foci found in the cortex.

Rabbit 54.—Weight 2,105 gm. Jan. 28: Test for albumin negative.

Jan. 31: Left kidney exposed and found normal. Injections of ascitic fluid as follows: Feb. 11, 0.5 c c; Feb. 19, 0.75 c c; Feb. 23, 1 c c; Feb. 28, 0.75 c c; Mar. 5, 0.75 c c; Mar. 12, 1 c c; Mar. 19, 1 c c; Mar. 26, 1 c c; Apr. 3, 1 c c.

Killed Apr. 8.

Albumin tests negative Feb. 14, 21, Mar. 2, 26, Apr. 5. Nine injections were given over a period of 56 days. No gross or microscopic lesions found in the kidneys.

Rabbit 65.—Weight 2,170 gm. Dec. 6: Albumin test negative. Left kidney exposed and found normal. Injections of

ascitic fluid as follows: Dec. 22, 0.25 c c; Dec. 26, 0.5 c.c; Dec. 31, 0.5 c c; Jan. 11, 0.75 c c; Jan. 22, 1 c c; Feb. 5, 1.5 c c (slight shock); Feb. 11, 1.5 c c; Feb. 19, 1.5 c c; Feb. 23, 2 c c; Feb. 28, 1 c c; Mar. 5, 1 c c; Mar. 12, 1 c c.

Killed March 18. Twelve injections were given over a period of 86 days. Albumin tests negative, Dec. 14, Jan. 3, 30, Mar. 2. No gross microscopic lesions found in the kidneys.

Experiments with Egg-Albumin.

Rabbit 44.—Weight 2,250 gm. Jan. 3: Test for albumin negative.

Jan. 5: Left kidney exposed and found normal.

Injections of a 1% solution of egg-albumin as follows: Jan. 15, 0.25 c c; Jan. 22, 0.5 c c; Jan. 26, 0.75 c c; Jan. 30, 1 c c; Feb. 5, 1.5 c c; Feb. 11, 2 c c of a 50% solution of egg-white intraperitoneally. Death, Feb. 11.

Albumin tests negative, Jan. 15, 25, 30. At necropsy the kidneys were found slightly swollen and moist. There was precipitated albumin in the capsular spaces and in the lumina of the convoluted tubes. Whether this albumin was excreted egg-albumin or serum-albumin was not determined. There were no areas of lymphocytic infiltration.

Rabbit 50.—Weight 1,850 gm. Feb. 28: Albumin test negative. Left kidney exposed and found normal. Intraperitoneal injections of a 50% solution of egg-albumin as follows: Mar. 14, 0.5 c c; Mar. 19, 1 c c; Mar. 26, 1 c c; Apr. 3, 1 c c; Apr. 8, 1 c c; Apr. 11, 1 c c; Apr. 16, 1 c c. Death, Apr. 19.

Albumin tests negative, Mar. 19, Apr. 5, 19. Seven injections were given during a period of 36 days. No gross or microscopic lesions found in the kidneys.

Rabbit 60.—Weight 1,560 gm. Dec. 5: Test for albumin negative.

Dec. 6: Left kidney exposed and found normal.

One c c of a 1% solution of egg-albumin was injected intravenously on the following dates: Dec. 20, 26, 31, Jan. 11, 22, and Feb. 5. One c c of a 50% solution of egg-white was injected intraperitoneally on Feb. 11, 19, 23, 25 and Mar. 5. Death, Mar. 9.

Albumin tests negative Dec. 14, Jan. 5, 15, 30, and Feb. 25. Trace of albumin, Jan. 25.

No gross changes found in the kidneys. Microscopically an occasional cast was observed, but there are no areas of lymphocytic infiltration.

Experiments With Sheep Serum.

Rabbit 38.—Weight 1,950 gm. Dec. 3: Albumin test negative. Dec. 6: Left kidney exposed and found normal. Injections of sheep serum, diluted with equal volume of salt solution, as follows: Jan. 19, 0.5 c c; Jan. 24, 0.75 c c; Jan. 29, 1 c c; Feb. 5, 1.5 c c. Found dead, Feb 6.

Albumin tests negative, Jan. 28, Feb. 1. The kidneys were swollen and cloudy. Precipitated albumin was found in the lumens of some of the convoluted tubules. There were no areas of lymphocytic infiltration.

Rabbit 42.—Weight 1,750 gm. Jan. 4: Test for Albumin negative. Jan. 15: Left kidney exposed and found normal. Jan. 19, 0.5 c c of sheep serum. Jan. 22, found dead. Kidneys were cloudy. No microscopic examination.

Rabbit 53.—Weight 2,970 gm. Feb. 7: Left kidney exposed and found normal. Feb. 14: Test for albumin negative. Injections of sheep serum as follows: Feb. 19, 0.5 c c; Feb. 23, 0.75 c c; Feb. 28, 0.5 c c; Mar. 5, 0.5 c c; Mar. 12, 0.5 c c; Mar. 21, 1 c c; Mar. 26, 1 c c; Apr. 3, 1 c c. Apr. 4, found dead.

Albumin tests negative, Mar. 12, 21. Kidneys showed no gross changes. Microscopically precipitated albumin was found in the lumina of many of the tubules. There were a few casts. No areas of lymphocytic infiltration seen.

Rabbit 63.—Weight 1,860 gm. Feb. 7; Test for albumin negative. Left kidney exposed and found normal. Injections of sheep serum as follows: Feb. 19, 0.5 c c; Feb. 23, 0.75 c c; Feb. 28, 0.5 c c; Mar. 5, 0.5 c c; Mar. 12, 0.5 c c; Mar. 21, 1 c c; Mar. 26, 1 c c; Apr. 3, 1 c c; Apr. 4, found dead.

Albumin tests negative, Feb. 14, Mar. 21. Trace of albumin, Mar. 12. Several small pits were found on the surfaces of each kidney. Microscopically there were small areas of lymphocytic infiltration corresponding to the surface pits.

Rabbit 67.—Weight 1,960 gm. Jan. 4: Test for albumin negative. Jan. 5: Left kidney exposed and found normal. Injections of sheep serum as follows: Jan. 19, 0.5 c c; Jan. 24, 0.75 c c; Feb. 5, 1 c c; Feb. 19, 1 c c; Feb. 23, 1.5 c c; Feb. 28, 1 c c; Mar. 5, 1 c c; Mar. 12, 1 c c; Mar. 21. 1 c c; Mar. 26, 1 c c; Apr. 3, 1 c c; Apr 11, 1 c c; Apr. 16, 1 c c; Apr. 18, 0.5 c c; Killed, Apr. 30.

Albumin tests negative, Jan. 17, 28, Feb. 25, Mar. 14; positive, Apr. 4, 18. A

number of fairly deep pits were seen on the surfaces of the kidneys: Microscopically rather extensive lymphocytic infiltrations were found underlying the pits. The lesions correspond with those of a well advanced spontaneous nephritis.

Rabbit 113.—Weight 1,824 gm. May 14: Albumin test negative. Left kidney removed and found normal grossly and microscopically. Injections of sheep serum as follows: May 28, 0.5 c c; June 4, 0.75 c c; June 13, 2 c c; June 27, 2 c c; June 29, 1 c c; July 2, 2 c c. Killed, July 11. Loss of weight during experiment, 474 gm. Albumin tests positive, June 27, July 2, 10. The right kidney was covered with pits. Rather large areas of lymphocytic infiltration were found deep in the cortex underlying the pits, the appearances being those of a spontaneous nephritis. Cultures from the kidney in dextrose broth gave a pure growth of a streptococcus.

Rabbit 103.—Weight 1,250 gm. May 2: No. albumin. Left kidney exposed and found normal. Injections of sheep serum as follows: May 28, 0.5 c c; June 4, 0.75 c c; June 6, 0.75 c c; June 13, 2 c c; June 27, 2 c c; June 29, 1 c c; July 2, 2 c. c. Killed, July 6. No loss of weight. Albumin tests positive, June 27, July 3. The kidneys showed many fairly deep pits. Microscopically there were rather large areas of lymphocytic infiltration. The appearances were those of a spontaneous nephritis. Cultures from the kidneys taken immediately after death gave a pure growth of a streptococcus.

Discussion.

These experiments indicate that ascitic fluid does not injure the kidney of a rabbit when injected intravenously in moderate amounts. The occasional small foci of lymphocytes found at necropsy in the injected animals can hardly be attributed to the proteins, since such lesions are commonly found in nonexperimental animals and were not excluded by the preliminary examination. Lesions of this size produce no changes on the kidney surface and seldom cause albuminuria.

The production of anaphylactic symptoms repeatedly did not cause nephritis. The symptoms were usually rather mild, but it is not possible to produce severe shock repeatedly. The animals either die or become refractory. The symptoms were apparently nearly as pronounced as in the experiments of Longcope and Boughton.

Egg-albumin in large doses seemed to cause a mild parenchymal injury of the kidney occasionally, but no chronic changes of any kind were produced. This protein seems to be only mildly toxic for rabbits.

Sheep serum, however, is evidently toxic for rabbits, especially in relatively large doses. Several animals died shortly after a single injection. It produces a tubular injury to the kidney, but this is not severe enough to cause the death of the animal. There is no evidence of special glomerular injury. Degenerative changes only were observed in the first three experiments; but during the summer inflammatory renal lesions were found in 4 rabbits. This is evidently the same lesion which Longcope obtained. It undoubtedly developed during the course of the experiments, since it was not present at the beginning. It is similar in all respects to ordinary spontaneous nephritis. Like the spontaneous disease it would seem to be due to a bacterial infection, since pure cultures of streptococci were obtained from the kidneys of rabbits 113 and 103.

Our interpretation of these apparently positive results with sheep serum is, therefore, that the animals acquired an infection during the course of the experiments. This infection may have been contracted from other rabbits in the animal house, or it may have been introduced by the intravenous injections, as these were not made with complete aseptic precautions. In either case it is probable that the injury of the kidney by the sheep serum favored the development of the infection. The injured kidney may have been a locus minoris resistentiae.

If this interpretation be correct it may be said that injections of sheep serum cause nephritis in rabbits by injuring

the kidney and giving opportunity for the lodgment and growth of bacteria which may gain access to the circulation; but this is not a nephritis due directly to the action of anaphylatoxin or foreign protein. It is conceivable that toxic foreign protein might have a similar effect in man, but it is unlikely that such proteins ever gain access to the circulation in such relatively large amounts as are used experimentally in animals. And finally, if we grant the possibility that toxic foreign protein may enter the circulation and injure the kidney, we would expect the development of lesions similar to those found in the rabbit. But the only human renal lesions comparable to spontaneous nephritis of the rabbit are the hematogenous infections, namely, abscess of the kidney, descending pyelonephritis, and acute interstitial nephritis. Aside from the arteriosclerotic kidney, nearly all cases of chronic nephritis in man are examples of chronic glomerulo nephritis which has no anatomic resemblance whatever to spontaneous nephritis in the rabbit. It is therefore clear that the experimental production of such a lesion as spontaneous nephritis does not throw any light on the etiology of chronic nephritis in man.

It is not improbable that the various experimenters, who believe they have produced a chronic nephritis experimentally with uranium, potassium chronic, foreign protein, streptococci, etc., have been dealing with a spontaneous nephritis which was contracted during the experiment and hastened in its development by renal irritants. But even if all these claims were true we still have made little progress on determining the etiology of chronic nephritis in man.

As regards the etiology of human chronic nephritis we share the view, now widely held, that streptococci are the main causative agents, and that they first produce acute glomerular lesions which in many instances heal, but in others pass into a chronic stage. The continuous escape of streptococci from a focal infection or thru a diseased mucous membrane would theoretically favor the development of chronic glomerular lesions. But thus far no one has been able to furnish experimental proof to support this theory.

Summary.

Nontoxic foreign proteins, such as ascitic fluid, do not produce renal injury of any kind in the rabbit when injected intravenously in moderate amounts. The repeated production of anaphylactic symptoms with this protein likewise does not injure the kidney.

Egg-albumin may produce slight parenchymal injury when injected in large amounts, but usually it causes no damage.

Toxic foreign proteins such as sheep serum produce considerable parenchymal injury to the kidney which in some instances seems to lead to the rapid development of a renal infection comparable in all anatomic respects to the ordinary type of spontaneous nephritis. The fact that streptococci were isolated in pure culture from the kidneys of such animals shows that the lesion is essentially infective, and not the direct result of foreign protein or anaphylatoxin.

No lesion comparable to human chronic glomerulo-nephritis has been produced experimentally.

There is no experimental evidence that foreign protein is in any way responsible for chronic nephritis in man.

USE OF THE ROENTGEN RAY IN THE DENTAL SCHOOL CLINIC FOR DIAGNOSIS IN GENERAL AND THE TREATMENT OF ROOT-CANALS.

Ray Roberts Knight, A. B., M. D., Minneapolis.

(Read before the American Institute of Dental Teachers at Atlanta, Georgia.)

IT IS an interesting commentary on the revolutionary changes taking place in dentistry today to note the ever increasing part which roentgenology plays on the programs of all meetings of the profession. Only a few years ago, the dental radiograph was considered an interesting experiment of rather limited practical value, tho general roentgenology had attained a permanent place in medicine. Now, the dentist who does not make constant use of this aid in diagnosis is considered a back number, even by the laity. The patient demands that uncertainty in dental diagnosis be decreased to the minimum. His reliance on the "X-ray," as he terms it, is more implicit than ours, for we, even in our most biased moments, realize that it has certain limitations. These limitations, however, form no excuse for failure to avail oneself of the very great benefits to be derived from the ray. It should be regarded, not as an all-sufficient diagnostic means, but as an exceedingly valuable addition to the armamentarium previously accumulated by the dentist.

The topic assigned me is exceedingly broad. It calls for consideration both of the ideal use of roentgenology in the dental clinic, and of the less extensive use which is actually made in most clinics for reasons of finance.

What draws people to a public dental clinic? The primary reason is of course, a desire to save money. This fact to quite an extent determines the general type of those who come. They comprise those who either cannot, or will not, spend much on their dentistry. Many of them have had bitter experiences with the "guarantee dentist", and the clinic offers them an opportunity to secure, at low cost, work done under the careful supervision of men who stand high in the profession. The necessity of holding down the cost is a large factor in determining the number of Roentgenograms to be used.

The practice in the College of Dentistry of the University of Minnesota is somewhat as follows: The patient on entering goes thru the hands of the examining department. Here is noted the general condition of the mouth, presence and degree of pyorrhea, presence and condition of pulpless teeth, crowns, bridges, cavities, etc. Many times the fistula from an old "gum boil" is evident, or a history of such trouble is easily obtained. The first object of the clinic is to clean up the mouth. Many of these cases come in an effort to seek out the cause of some systemic disturbance, such as neuralgia, rheumatism, cardiac or gastric trouble. Those teeth which obviously are not worth repair are noted for extraction, and the instructor orders rayed all crowns, bridges, and devitalized teeth with large fillings which appear as if they might have gradually lost vitality from injury to the pulp, and to teeth which may have died as the

result of a blow. All work, except the extractions already mentioned, is held in abeyance till the findings of the Roentgenograms are available. The plans of mouth restoration, or the advisability of a complete cleanup, with subsequent plates, can now be worked out with some scientific exactness. The pyorrhea patients do not as a routine pass thru our hands. There are those cases in which there is some doubt as to the degree of destruction of the alveolar process, and these we ray. Occasionally it is desired to show what bone re-formation, if any, has taken place after treatment. Often in raying for other reasons, we show an unsuspected pyorrheal absorption around teeth which had appeared sound. There are instances of teeth responding to all tests for vitality, and yet showing very definite tip rarefactions.

The Orthodontia Department uses us to quite an extent to determine the presence and position of unerrupted teeth. As to their position, we meet with one of our greatest difficulties. The Roentgenogram is an attempt to secure on a flat surface a shadow gram of a series of curved objects. To do this without some exaggerations of perspective is plainly impossible. If you correct the exaggeration in one direction, you render it worse in another. Therefore, you direct the ray perpendicular to the approximate center of the mean curve of the process, and so compromise the matter. In reading the film, you make mental allowance for this fact. The result is not altogether satisfactory to the orthodontist, leaving somewhat in doubt the real relative positions of the visible and unerrupted teeth. Of course, added information may be obtained by making two films with a shift of the tube, as in stereo work. It is also sometimes worth while to make films with the ray directed at angles which produce a poor picture except in some one particular.

Certain cases intended for extraction are first sent to the Roentgen Department. These include, chiefly the impacted third molars, and teeth previously broken off, where it is desirable to know the number and position of root fragments left in the process. We sometimes are asked to locate broken needles, and other impedimenta not desirable as permanent fixtures.

The use of roentgenograms in our clinic in connection with root-canal work is more limited than we would wish. Here the cost is relatively high as compared with the treatment, and it is difficult to persuade the patient to pay the extra amount for what seems to him an intangible gain. Those cases are rayed in which difficulties develop during the work of canal filling, or in which there is doubt as to the number, direction and potency of the canals.

Our ideal for the use of the ray in the clinic would not differ so widely from what has already been outlined. The ideal in dentistry today is to place the entire mouth in as good a condition as possible, rather than only to stop the pain which brings the patient. The public is gradually being educated along this line,—the idea of restoring and reconstructing the mouth. Such an ideal has for some time been carried out by many men in their private practices, and it is in process of attainment in our public clinics. This involves a detailed examination of the entire mouth, including roentgenograms of those teeth which fall under suspicion. The safest procedure is to ray all the teeth, for we are often surprised by finding pathology on teeth which gave no other evidence of trouble. This fact sometimes gives rise to spirited discussion as to whether the roentgen testimony is reliable. With the findings of the examination and of the films at hand, an intelligent outline of restoration can be made. During the process of such work, the ray should be employed at various stages as a check-up on thoroness. Such a routine would

make the student much more careful in his canal work. Attempts at measuring canal lengths by raying with wires in the canal have been rather disappointing, so far as actual net results are concerned. The measuring can be accomplished with very fair accuracy, but to apply the information so as to secure ideal canal fillings involves difficulties familiar to you all. During the last few years much time has been spent in discussing—and cussing the "root-canal problem". My own opinion is that the next few years will see the elimination of this problem by the elimination of the pulpless tooth. The pulpless tooth, no matter how disguised by verbiage, is a foreign body in the alveolar process. Nature dislikes the presence of such a foreign body and no amount of therapeutic "kidding" can convince her that it is right.

We have had the opportunity of making a number of series of roentgenograms of cases following apicoectomy—one of the fads of recent years. They are exceedingly interesting—as pictures. From month to month the film shows increasing bone re-formation, till the area seems almost like the original process. Basing our judgment on these findings, we grew rather enthusiastic over this surgical procedure. Then cases began to drift back with a complaint of returned and increasing soreness, and another idol was shattered. The infection had not been really eliminated.

And now, in conclusion, we reiterate our early statements. Roentgenology has made a very valuable contribution to dental science in the last few years. It has become indispensible to the careful and conscientious worker, not as a substitute for his other methods of diagnosis, but as an aid to them. Its evidence should be correlated with theirs to reach an accurate result. And its use in the public clinic, already extensive, should be increased, to assist the other departments in their tireless effort to give the clinical patient the very best in dentistry.

GOLDEN ANNIVERSARY IN DENTISTRY.

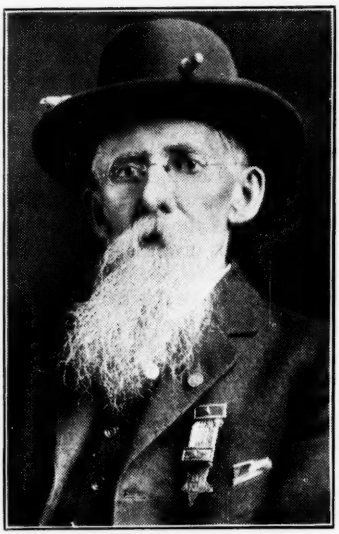
By J. P. Luthringer, D. D. S., Peoria, Ill.

THE accompanying cut is a good likeness of Samuel Alexander Campbell, of Mattoon, Illinois, whose golden anniversary in the active practice of dentistry was celebrated on November 18th, 1919, with the Eastern Illinois Component Society as the congratulants. Over a hundred were seated at the banquet tables, comprising the members of the society, local friends, and fellow members of the Illinois State Dental Society. Among the latter were four past presidents, viz: J. G. Reid, C. N. Johnson, D. M. Gallie and J. E. Hinkins, all of Chicago.

Dr. Gallie was the toastmaster, acquitted himself, as usual, in the most satisfying manner. Among others he introduced the following men who responded to toasts. Senator A. C. Clark, Chicago, spoke entertainingly of his boyhood days in Mattoon, and likewise of his thrilling experiences in making tests for the government during the war in the aerial service; G. H. Damron, Arcola, spoke on "Dr. Campbell as My Preceptor; "J. P. Luthringer, Peoria, State Secretary, spoke on "Like Members"; C. N. Johnson, Chicago, paid a fine tribute to Dr. Campbell in his remarks on "Fifty Years a Servant of Mankind."

As a fitting climax to the evening's festivities, a silver loving cup was presented to the honored guest by the Eastern Illinois Society. Dr. Campbell, occupying the seat of honor at the table, smiled bravely thru his tears while his friends extolled his services as a soldier, patriot, and benefactor to his community for over a half hundred years. In a few well chosen phrases Dr. Campbell expressed his gratitude for the honors that were showered upon him.

With the exception of a short interval, during which time he was a casual, Dr. Campbell served thruout the civil war. He then took up the study of dentistry at the Dental College of Philadelphia, graduating in 1869. Shortly thereafter he established an office in Mattoon, and



SAMUEL ALEXANDER CAMPBELL.

at the age of 71 is still actively engaged in the practice of dentistry in his adopted city.

Dr. Campbell is an honor to the profession, to the society, and to his city. He has always stood for the best there was in the profession, and out of it, and the encomiums of his friends fall in benediction upon a man, who during more than three score and ten years of life, has thought right, lived right and done right.

CARR PATENT INVALID.

Dental Protective Association of the United States Wins Important Decision.

ON THE afternoon of December 15, 1919, the Dental Protective Association of the United States held its annual meeting at the Palmer House, Chicago. Dr. J. G. Reid was reelected President, Dr. Donald M. Gallie, Treasurer, and Dr. Elmore Elliott, Secretary of the organization.

The principal matter of business which came before the Association was in regard to the litigation undertaken by it some three or four years ago in connection with the patent to Cassius M. Carr on pyorrhea scalers, No. 1,138,355 of May 4, 1915. While Carr brought numerous suits under this patent, the one against Dr. Austin F. James, of Chicago, defended by the Dental Protective Association of the United States, was the only one ever brought to trial, and resulted in a decree, affirmed by the higher court, holding the Carr patent void.

Inasmuch as this litigation and its outcome are of probably greater interest than any other litigation in a great many years to thousands of dentists all over the country, we are giving below extracts from the remarks of the attornies of the Association upon this subject at the annual meeting referred to.—Editor.

Mr. Percy B. Eckart, of Chicago, General Council for the Association, who was in charge of the defense of the suit on behalf of the Association, in making his annual report of the outcome of the litigation, said in part:

On Tuesday, October 7, 1919, the United States Circuit Court of Appeals for the Seventh Judicial Circuit, comprising Judges Bakers, of Indiana, Evans, of Wisconsin, and Page, of Illinois, sitting at Chicago, Ill., nois, handed down a decision and opinion affirming the decision of Judge Carpenter, of the United States District Court, who decided in June, 1918, that the patient of Cassius M. Carr, which he claimed to cover a set of 150 tools for the treatment of pyorrhea was void, and ordering the dismissal for want of equality, of the bill for injunction and damages for infringement and unfair competition which The Carr School of Preventive Dentistry and Medicine had brought against Dr. Austin F. James. This suit has been pending in the United States Courts for over four years.

Briefly summarizing, I may remark that Cassius M. Carr obtained from the United States Patent Office, after ten years of persistent effort, a patent on a set of dental instruments which he claimed were novel and patentable. Dr. James had bought instruments manufactured by the Hartzells, of Minneapolis, similar to the Carr set, embodying a principle that has been recognized in dentistry for a great many years. Carr thereupon filed a bill for an injunction and claimed an infringement of his patent and unfair competition against both James and the Hartzells. The Dental Protective Association believed that a service might be performed to the whole dental profession if this

alleged patent of Carr's were shown to be void and unenforceable because the tools he claimed to have invented were old and otherwise not patentable, and undertook the defense of both of these suits. We obtained the dismissal of the Hartzell suit a year or more ago.

The testimony of more than sixty witnesses was taken in the James case, the hearings covering a period of over two years' time, and occurring in New York, Philadelphia, Minneapolis and Chicago. The parties to the suit and many prominent practitioners and experts testified for Dr. James. Carr's witnesses comprised largely his former pupils and his past or present associaties. After all the evidence was taken the case was argued in June, 1918, before Judge Carpenter and resulted in a complete victory for Dr. James and the Dental Protective Association of the United States in that the patent of Carr and the Carr School was declared void and the suit ordered dismissed for want of equity. The costs of the court proceedings were assessed against Carr. Carr thereupon took an appeal to the United States Circuit Court of Appeals. The case was argued last spring before these three judges and printed briefs were filed. The decision of the lower court was affirmed. This finally ends Carr's patent in this jurisdiction at least, and the Association is now in possession of all the facts, documents and necessary exhibits which would conclusively defeat any further suits should Carr undertake to prosecute any member of the Association in any other part of the United States.

The decision is of great importance to the Association and to the dental profession as a whole, because so long as the Carr patent remained unassailed there was a possibility of widespread harrassment of the profession by Carr with patent infringement suits and unfair competition proceedings.

The claims of his patent were broad. Almost any dental tool which had the two-point contact principle or had the cutting edge on line with the longitudinal axis of the handle and was operated with a drawing motion, might be claimed an infringement. Carr seemed to assume that he was inventor of tools in a series for the treatment of pyorrhea and the discovery of the method of removing calcareous deposits from teeth without injury to the surrounding tissues by the so-called planing action of the tools.

The Association's attorneys showed by overwhelming proof that the patent office had been misled into granting the patent at all. That is was void for scores of reasons. Untrue description and ambiguity in the language of the patent, the utter impossibility of construction of the instrument described and especially the lack of any novelty whatsoever in the alleged invention.

It was shown that the pyorrhea tools had been anticipated many years by sets of instruments used and manufactured by Drs. Cravens, MaWhinney, Tompkins, Johnson, Jones and others and that the Carr set followed almost identically a description published by Dr. G. V. Black in the Dental Digest in 1898.

This litigation brought by Carr under the name of the Carr School set against an individual dentist proved to be quite expensive for both parties, in time and in money. Fortunately for Dr. James, his membership in the Dental Protective Association of the United States and that Association's assumption of the defense of the suit relieved James of the financial burden which would be, in any case of this kind, too much for any individual.

This case, therefore, convincingly demonstrates the advantage of the Assocation to its members, who, for a nominal sum, may secure insurance and immunity against great financial loss in case of an attack by a person such as Carr capitalizing a mere paper patent.

Dentists belong to that class of per-

sons peculiarly subject to attack under patents, because of the large number and variety of the instruments and processes which they habitually employ in their professional work, and the fact that many patents have been issued and are constantly being issued for instruments and processes used by the profession.

The sole purpose of the Dental Protective Association of the United States, which has been in existing and active service to its members for more than thirty years, is to determine whether a patent upon an instrument or process of general interest to the profession is invalid and should be defeated as a matter of justice and right, and in case of suit on the patent against a member of the Association, to defend that suit, paying all court costs and attorneys' fees in connection with it.

Any dentist not now a member of this Association is missing an opportunity not only to save himself a large sum of money and loss of time in patent litigation, which may come upon him unexpectedly at any time, but a most excellent opportunity to cooperate with thousands of his fellow practitioners who are members of the Association, which has been doing a splendid work in upholding the fundamental right of dentists.

Mr. Luther Johns, of Chicago, Illinois, patent lawyer, associated with Mr. Eckert in the Carr case, spoke in part as follows:

"This Carr patent has been a focal infection in the dental body. In Chicago the inflammation has been severe. Until recently the Carr school was located here, and here has been the battle ground to test the validity of the patent.

"I am satisfied that some of the Carr students consider Carr in the light of a pioneer in the treatment of pyorrhea. Of course, as you gentlemen know, and as I learned from my study of the history of pyorrhea treatment in connection with this case, there has not been anything fundamentally new in the treatment of pyorrhea by instrumentation within the last fifty years. There has been improvement in some details of the practice and there is a larger proportion of dentists today who have acquired the necessary understanding as well as the required skill of manipulation, but if you will turn to the text books and the papers read before dental societies and the discussions of them reported in the earliest dental magazines, as I have done, you will find that in those days the essentials in the treatment of pyorrhea were fully understood. Cures were then effected by the identical methods relied upon today.

"I mention these facts as a preamble to the observation that when Carr's students speak of Carr as the *alpha*, and sometimes also as the *omega* of the art of oral prophylaxis, as they sometimes do, they merely convict themselves of unacquaintance with the history of the subject, for oral prophylaxis, as a system of hygiene, was understood and practiced, and by substantially the same methods as are employed today, at least as early as the times I have already mentioned.

"This leads to the further observation that I am sure a number of these Carr students, including some estimable members of this Association, while not criticizing the Association for the part it took in defeating the Carr patent, are inclined to entertain a feeling that in the defeat of this patent something has been taken from Carr which was his own. I base this upon the attitude of some of the Carr witnesses and some conversations I have had with other dentists, including one who expressed himself rather forcefully along this line.

"I may say in this connection that the Association, thru its attorneys, never minimized the value of anything that Carr really did. We stated frankly to the court, and everywhere else when occasion suggested it, the value of a larger set of instruments over a smaller

set. Dr. Hartzell frankly paid Carr a tribute along this line upon the record of the case. Such an accomplishment, however, does not involve invention. Thru the exercise of patience and a pair of pliers used for twisting the bits or bending the shanks, a larger variety of such tools, vastly greater in number than even the Carr set, can readily be made from a single starting point, for instance one of the Cravens, MaWhinney, Tompkins, and many others of the prior art. The utility of a larger set of pyorrhea scalers over a smaller set is strictly comparable to the utility of a larger set of pluggers over a smaller set, and some sets of these pluggers run in numbers upwards of five hundred.

"It does not necessarily follow that because a thing has utility it has any proper relation to the patent law. Indeed, of all the manifold things of utility and advantage in life only on exceedingly small percentage of them has any proper relation to the patent law.

"Not one of these estimable gentlemen who may conceive that Carr has been deprived of some right thru the defeat of his patent would insist for a moment that he should have a patent if he were not entitled to it. Now our federal trial court and Court of Appeals have both decided that Carr was not entitled to the patent. And these decisions were reached, not over night, but after more than three years of preparation in which an enormous number of witnesses were examined, in which at least four different groups of attorneys cooperated with Carr to present his case, and in which the hearings were upon full oral argument and written briefs.

"A patent is defined and limited by its claims. Carr's patent had two claims, the first calling for a single tool, and the second calling for a series of tools. It is obvious that altho a dentist should take the Carr course and learn enough about the principles of cleaning teeth thru the manipulation of instruments to enable him to assume that he is now a pyorrhea specialist, there is not necessarily the slightest relation between that fact and the validity of a patent claiming a monopoly on any one or any two or more of the tools exployed. No one would contend that because a medical student learned the art of surgery from a certain surgeon, that surgeon ought to have a patent covering one or two of the old forms of scalpels used.

"The highest court to which Carr can appeal has decided that Carr was not entitled to a monopoly on the subject matter of his patent claims, and has said in effect, as the judgement defeating any patent says in effect, that to continue the patent in force would be a fraud upon public rights. It is obvious, that any sympathy for Carr in the loss of his patent is due either to a failure to understand the significance of a court decision or else to a mere maudlin sentimentality.

"The fact is that Carr claimed as his own that which he was not entitled to claim as his. His patent purported to give him the right to make, use and sell exclusively dental instruments which every dentist in the country has had the right to make, use and sell from the beginning of the dental art.

"Everyone, therefore, who is able to look at a matter fairly, and who has a just regard for the integrity of the patent system and for the public right, must rejoice in the defeat of this exceedingly obnoxious grant, and must give unstinted praise to the Dental Protective Association of the United States for its successful work in this behalf."

THE JOURNAL of the NATIONAL DENTAL ASSOCIATION

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Editorial Department.

TWO DEANS.

Within a few days of each other, two men who had acted for years as deans of dental colleges passed away—Dr. Henry W. Morgan, of Nashville, Tenn., on January 17th, and Dr. B. Holly Smith, of Baltimore, Md., on January 22nd, 1920. Dr. Morgan had been Dean of the Dental Department of Vanderbilt University from 1911 till forced to retire owing to ill health in June, 1919. Dr. Smith was dean of the Baltimore College of Dental Surgery, and had given the best years of his life to the service of this institution.

It is well to pause in an event of this kind and consider something of the sacrifice that men in our profession are making for the cause of

dental education. Both of these men carried burdens that the rank and file of the profession little dream of, and both have probably died before their time with these burdens as a contributing factor. It is so easy for men who are not engaged in dental teaching to criticise those who are—it is so easy to find fault, to censure, to disparage. It is easier to do this than to say the kindly encouraging word which would help so much over the hard places that occur with such great frequency in this exacting pursuit. Few men not engaged in dental teaching have the slightest conception of the difficulties to be encountered or the problems to be solved. No dean of a dental college ever sleeps so soundly or so tranquilly as he would without this responsibility, and few deans ever go out to seek for the position they hold. Usually the place is thrust upon them because their predecessor is worn out and wearied with the burden, or else has died, and yet many of them have been classed as self-seekers and glory hunters.

Small reward this, for the men who have unselfishly devoted the best energies of their lives that the young men entering the profession may have the benefit of a well rounded dental education. It has been the prevailing fad or fashion for years to criticise dental education, as if those engaged in this work were a lot of vampires preying on the unsuspecting profession for their own aggrandizement. Most of this criticism has come from men who have never done any teaching and who are unfamiliar with the ceaseless problems presented in this field of effort. If they only knew!

Doubtless many reforms have been needed in the past, doubtless many are needed today, and many will be needed as long as dental education continues. No one is more conscious of this than are those who are engaged in the work—no one is more desirous of bringing about reform. But a constant carping criticism does not tend toward a reform —it only wears out the lives and distracts the energies of those who are placed in these positions of responsibility.

When we stop to study the personnel of the men who have acted as deans of the dental colleges of this country we are impressed with the sterling character of the majority of them. Many of them have passed to their reward, leaving behind them a record of unselfish devotion. Beautiful tributes have been paid to them after their death, when all too frequently their acts while living had been subjected to unwarranted censure. Why cannot we say the kindly and encouraging thing to men while they are still alive? Why wait till they are cold and unresponsive to seek for the good there is in them?

Drs. Morgan and Smith, in their capacity as deans probably escaped criticism more generally than most men in their position, and

yet we venture the opinion that the full meed of praise was too frequently withheld from them, at times when praise would have gone so far toward making their pathway more pleasant.

The passing of our prominent men marks an ever changing era in the profession, and we trust we are not indulging a vain hope when we bespeak for the eras to come a larger charity and a more abounding tolerence for the difficulties and the limitations of those who are called upon to occupy positions of trust and responsibility. Let us look for the good there is in men, and we will have less incentive and less relish to magnify their faults. Then our great men will live longer and be happier.

THE DENTAL PROFESSION'S TEN AIMS FOR 1920.

1. 29,000 members and 35,000 subscribers to The Journal.—Average of 18% increase in membership in every component society.

2. $78,000 in the Relief Fund with a definite plan of distribution.—Every member a contributor to this worthy cause.

3. Thru the Research Commission and otherwise encouraging the spirit of scientific research and inventive genius.—Debt on the Research Institute Building paid.

4. 6,000 in attendance at the Boston meeting and the staging of a real "Seminar Course."

5. 2,000 additional volumes added to our National library.

6. Systematic and standardized graduate and post-graduate courses inaugurated and developed.—The educational standard in every dental school raised.—The classification of State Dental Examining Boards.

7. More efficient dental service to humanity.—National Oral Hygiene Bureaus established.—More public dental clinics.—Better dental laws.—Dental Hygienists recognized in every state.

8. All scientific papers to include complete bibliography.—Revision and standardization of nomenclature.

9. The three requisites for the successful dentist: Enthusiasm, Energy and Thoroness.

10. For every dentist to have a religion of truth, law, justice, work, democracy, mercy, monogamy, science and God.

NATIONAL DENTAL ASSOCIATION, 1920.

August.

23-24-25-26-27—Boston.

April.

5-7—American Society of Orthodontia, Chicago, Ill.

STATE SOCIETIES.

March.

4-5-6—Minnesota, Minneapolis.
8-9-10-11-12-13—Texas, Dallas.
15-16-17-18-19-20—Oklahoma, Tulsa.
17-18-19—Vermont, Burlington.
22-23-24-25—Illinois, Chicago.
22-23-24-25-26-27—Kansas, Wichita.
29-30-31, April 1-2-3, South Dakota, Sioux Falls.

April.

7-8-9—Kentucky, Louisville.
12-13-14—Michigan, Detroit.
12-13-14—Missouri, Kansas City.
13—Alabama, Mobile.
14-15-16—West Virginia, Charleston.
19-20-21-22-23—Mississippi, Jackson.
20-21-22—Connecticut, New London.
27-28-29—Pennsylvania, Reading.

May,

Massachusetts, Boston.
North Dakota, Grand Forks.
4-5-6—Iowa, Cedar Rapids.
13-14-15—New York, Albany.
17-18-19-20-21-22—Nebraska, Lincoln.
18-19-20—Indiana, Indianapolis.

June.

Florida, Miami.
Maryland, Baltimore.
Maine.
New Hampshire, Manchester.
New Mexico, Albuquerque.
Southern California, Los Angeles.
South Carolina, Columbia.
Utah, Provo.
9-10-11—Georgia, Atlanta.
14—Colorado, Colorado Springs.
14-15—Wyoming, Cheyenne.
North Carolina, Wrightsville Beach.

July.

New Jersey.
Virginia, Old Point Comfort.
13-14-15—Wisconsin, Milwaukee.

November.

18—District of Columbia, George Washington University, Washington, D. C.
Arizona, Phoenix.

December.

2-3-4—Ohio, Columbus.
10—Delaware, Wilmington.

Dates to Be Announced.

Arkansas, Little Rock.
California State.
Idaho, Pocatello.
Louisiana, New Orleans.
Montana, Great Falls.
Oregon, Portland.
Tennessee, Chattanooga.
Washington, Tacoma.

ILLINOIS STATE DENTAL SOCIETY.

The fifty-sixth annual meeting of the Illinois State Dental Society will be held in the Congress Hotel, Chicago, March 22, 23, 24 and 25, 1920.

The following essayists have been secured: Dr. Alfred C. Fones, Bridgeport, Conn.; Dr. Charles S. Essig, Philadelphia, Pa.; Dr. Wm. H. G. Logan, Chicago; Dr. Newton G. Thomas, Chicago; Dr. Hart J. Goslee, Chicago; Dr. F. E. Roach, Chicago; Dr. K. W. Knapp, Des Moines, Iowa; D. A. A. Herdliska, United States Public Health Service, Washington, D. C.

A most cordial invitation is extended to all ethical practitioners in Illinois and neighboring states.

G. WALTER DITTMAR, Pres.
J. P. LUTHRINGER, Secy.

OKLAHOMA STATE DENTAL SOCIETY

The next meeting of the Oklahoma State Dental Society will be held in Tulsa, Oklahoma, March 15-16-17-18-19-20, 1920 This meeting will be conducted on the class system of post graduate instruction.

The attendance will be divided into classes, by enrollment previous to the time of the meeting and each class will take up the intensive study of one subject for the full week.

At this time the following teachers have been engaged:

Dr. Thos. B. Hartzell, Pyorrhea and Prophylaxis.

Dr. Arthur E. Smith, Nerve Blocking and Anesthesia.

Dr. Dayton D. Campbell, Full Dentures.

Dr. F. E. Roach, Partial Removable Dentures.

Dr. T. W. Maves, Inlays and Abutments for Vital Teeth.

These teachers will have assistants to help with the class work. The instruction will consist of lectures, technical and practical demonstrations. Ample facilities will be provided for clinical instruction and where possible the students of each class will be expected to do much of the practical work.

This meeting is one of a chain of meetings to be conducted by the Texas, Kansas, South Dakota and Oklahoma State Dental Societies. Any dentist, in good standing in his state society with 1920 dues paid, desiring to attend this meeting will be permitted to do so, provided the class of his choice is not filled by our own members. The class fee is fifty dollars.

The Bulletin of the Oklahoma State Dental Society, which gives full information about our meeting will be mailed to any one upon request.

C. R. LAWRENCE,
General Manager,
Stephenson Bldg., Enid, Okla.

PENNSYLVANIA STATE DENTAL SOCIETY.

The fifty-second annual meeting of the Pennsylvania State Dental Society will be held in the Rajah Theatre, Reading, Pa., April 27, 28 and 29, 1920. An interesting and instructive program has been prepared. All ethical practitioners are invited to be present.

W. L. FICKES, Sec'y,
6004 Penn Ave., Pittsburgh, Pa.

HOME COMING, ALUMNI ASSOCIATION, WASHINGTON UNIVERSITY.

Are you planning to attend the Home Coming of the Alumni Association of Washington University School of Dentistry, February 23-24? There will be practical demonstrations as well as individual clinics and clinics of the St. Louis Study Club. This meeting promises to be the best and most interesting ever held, so help yourself and the Alumni Association by being present.

A. VERNETTE SANDUS,
Publication Committee, St. Louis, Mo.

SOUTH DAKOTA STATE DENTAL SOCIETY.

The 38th annual meeting of the South Dakota State Dental Society will be held in Sioux Falls, March 29 to April 3, 1920. One week of post graduate instruction will be given under the following clinicians:

Dr. Arthur E. Smith, Chicago, Ill.

Dr. F. Ewing Roach, Chicago, Ill.

Dr. Dayton Dunbar Campbell, Kansas City, Mo.

Dr. T. W. Maves, Minneapolis, Minn.

ERNEST W. ELMEN, Secretary,
608 S. F. National Bank Bldg.,
Sious Falls.

MASSACHUSETTS STATE BOARD OF DENTAL EXAMINERS.

The next meeting of the Massachusetts State Board of Dental Examiners will be held in Boston, Mass., March 10th, 11th, 12th and 13th, 1920, beginning promptly at 9:30 A. M., March 10th.

All applications must be in the hands of the Secretary by March 1st. Fee for examination, $20.00. Full information and application blanks may be received by addressing

J. N. CARRIERE, D. D. S., Secy.,
352 Main Street, Fitchburg, Mass.

TWENTY-FOURTH ANNUAL MEETING

National Dental Association, Boston, Mass., August 23-27, 1920.

"Listen my children and you shall hear,
Of the Midnight Ride of Paul Revere."

So runs the masterful poem of Henry Wadsworth Longfellow that has so thrilled us in our school days, but you say what has this to do with dentistry?

Paul Revere organized, and was elected First President of the Massachusetts Mechanics Association and this association erected the famous Mechanics Building, which is known all over the continent as the home in Boston, Mass., of the far famed poultry shows, automobile and dog shows, mechanics and food fairs, and athletic meets.

Massachusetts Mechanics Building is 600 feet in length, covers three acres of land, and contains over 150,000 square feet of floor space, being one of the largest buildings in the United States for exhibition purposes of every description.

August 23-24-25-26-27, 1920, the Mechanics Building Boston, Mass., will be the home of the National Dental Association, conducting the biggest, busiest, and best dental meeting ever held in the history of dentistry. The many halls on the second floor, make it ideal for a Seminar Course meeting, which is the plan of the committee for this year. While the almost unlimited floor space of the grand exhibition hall on the main floor, will afford ample room that all manufacturers may exhibit to any extent they desire, and assures the finest exhibit of dental materials and specialties, ever gathered together under one roof in this or any other country.

This building contains many relics of historic interest to all who will attend this meeting, and the committee in charge is all ready at work, making arrangements to care for six thousand members of the profession, while the entertainment committee has mapped out a program for the entertainment of the ladies, exceeding anything ever before attempted at the National Dental Association meetings.

F. E. JEFFREY, Chairman,
Publicity Committee National Dental Association.

VERMONT STATE DENTAL SOCIETY.

The Vermont State Dental Society will hold its next annual meeting in Burlington on Wednesday, Thursday and Friday, March 17 to 19, 1920.

PHILIP E. MELLEN, Secretary,
Middlebury, Vermont.

CHICAGO DAY, TUESDAY, MARCH 23, 1920.

During the Illinois State Dental Society Meeting.

The Illinois State Dental Society's annual meeting will be held in the Congress Hotel, Chicago, Illinois, March 22-25, 1920. Tuesday, March 23, has been given over to the Chicago Dental Society and will be known as CHICAGO DAY. The program for this day will be under the auspices of the officers of the Chicago Dental Society. The entire day, both forenoon and afternoon, will be devoted to clinics, while the evening program will consist of a banquet in honor of Dr. Thomas L. Gilmer. Some new plans will be inaugurated in staging the clinic.

Ten Sectional Unit Clinics.

1. There will be ten sectional unit clinics with forty clinicians. The clinics will be given in the nature of a lecture and demonstration on the live subjects before the dental profession of today.

2. Two clinicians from each sectional unit will lecture and demonstrate in the morning and a like number will follow in the afternoon. However, each clinician will lecture and demonstrate his own particular technic.

3. The forty clinicians that have been invited to participate in these clinics are men of national reputation in the field they are to demonstrate.

4. Ample space has been secured so that everyone may have an opportunity to see this novel clinic manned by such an unusual galaxy of talent.

5. The only clinic given during the annual meeting of the Illinois State Dental Society will be given by the Chicago Dental Society on Tuesday, March 23.

Banquet to Thomas L. Gilmer.

The program committee of the Chicago Dental Society has arranged to give to Dr. Thomas L. Gilmer a testimonial banquet. This banquet will be held on Tuesday evening—CHICAGO DAY—in the Gold Room of the Congress Hotel. Men of national and international reputation have accepted invitations to attend this meeting and participate in the literary program. The program committee announces the following:

Dr. Edw. T. Darby, Philadelphia, Pa.
Dr. A. W. Thornton, Montreal, Canada.
Dr. John V. Conzett, Dubuque, Iowa.
Dr. Thos. P. Hinman, Atlanta, Ga.
Dr. Truman W. Brophy, Chicago, Ill.
Dr. Frank Billings, Chicago, Ill.
Dr. H. E. Friesell, Pittsburgh, Pa.
Dr. Arthur D. Black, Chicago, Ill.
Dr. G. Walter Dittmar, Chicago, Ill.
Dr. C. N. Johnson, Chicago, Ill.
Dr. Frederick B. Noyes, Chicago, Ill.
Dr. H. J. Burkhart, Rochester, N. Y.

The officers of the Chicago Dental Society are putting forth every effort to make CHICAGO DAY stand out in bold relief as one of the big days in the history of dentistry in Illinois.

VICTOR H. FUQUA, President,
25 E. Washington St., Chicago, Ill.

ALUMNI SOCIETY OF THE DEWEY SCHOOL OF ORTHODONTIA.

The next annual meeting of this society will be held on April 1-2-3, 1920, at the Edgewater Beach Hotel in Chicago.

The usual high standard of the meetings of this Society will be maintained. One half day will be devoted to clinics.

All interested in Orthodontia are cordially invited to attend these meetings.

GEORGE F. BURKE, Secretary.
741-45 David Whitney Bldg.
Detroit, Michigan.

MINNESOTA STATE DENTAL ASSOCIATION.

The thirty-seventh annual convention of the Minnesota State Dental Association will be held at the West Hotel, Minneapolis, Minn., on March 4th, 5th and 6th, 1920. MAX E. ERNST, Secretary,
1125 Lowry Bldg., St. Paul, Minn.

ANNUAL CONVENTION OF TEXAS STATE DENTAL SOCIETY.

The Texas State Dental Society will hold its 40th annual convention on March 8-13, 1920, at Dallas, Texas. The special feature of this meeting will be post-graduate class courses conducted on the "Oklahoma Plan." These classes will be conducted by the following well known men: Dr. Thomas B. Hartzell, Minneapolis, Minn.; Dr. F. Ewing Roach, Chicago, Ill.; Dr. Arthur E. Smith, Chicago, Ill.; Dr. Dayton D. Campbell, Kansas City, Mo.; Dr. T. W. Maves, Minneapolis, Minn. Other instructors may be employed.

J. G. FIFE, Secretary,
736 Wilson Bldg., Dallas, Texas.

MISSISSIPPI DENTAL ASSOCIATION.

The Mississippi Dental Association will hold its annual meeting at Jackson, April 19-23, 1920, inclusive.

W. F. BEAVERS, Secretary
Meridian, Miss.

NOTICE.

Twelfth Annual Congress of the American School Hygiene Association to be Held in Cleveland February 24-25-26-27-28.

The preliminary program of the 12th congress of the American School Hygiene Association to be held in Cleveland the latter part of February, has just been issued by its Secretary, Dr. William A. Howe, of the State Department of Education, Albany, N. Y.

This association is composed of prominent educators and health workers in various parts of America. The program as outlined will consist of nine one-half day sessions, beginning on Tuesday morning, February 24, and closing on Saturday morning, February 28.

All of the first day will be devoted to the consideration of Health Education. The entire second day will be devoted to School Health Service, including School Medical Inspection, Physical Education, School Nursing, and other activities under the general title of school health service. The session on Thursday morning will be devoted to Sex Education and Communicable Diseases. State Commissioner of Health, Freeman, of Ohio, is listed as chairman of this section. Thursday afternoon is given to the subject of nutrition of school children under the chairmanship of Dr. William R. P. Emerson, of Boston. On Friday morning seven papers on "Mouth Hygiene" will be presented by prominent dentists of this country and Canada. The full program of this section is given below. Friday afternoon will be devoted to Mental Hygiene under the Chairmanship of Dr. Henry H. Goddard, of Columbus, Ohio. The closing session on Saturday morning will be devoted to a symposium on the Relation of Health and School Authorities. Dr. Willard S. Small, of the U. S. Bureau of Education, is chairman of this section.

The entire program is filled with much valuable material and will prove, in our opinion, the most constructive and progressive contribution of recent years to our literature of School Health Service.

It is earnestly hoped that the dental profession of the country will avail itself of the opportunity, not only to hear the section devoted to Mouth Hygiene, but many others of the congress.

Membership in the association, which includes a copy of the proceedings of the congress, is $3.00 Let the Secretary hear from you.

Friday Morning, 9:30.

Mouth Hygiene.

How can the National Dental Association aid in a program for better teeth and better health for school children?

Otto U. King, D. D. S., Secretary
National Dental Association.

Teaching of Mouth Hygiene to School Children from the viewpoint of the Dental Journalist.
Rea P. McGee, M. D., D. D. S., Editor Oral Hygiene, Pittsburgh.

The relation of good teeth, clean mouths, and good nutrition to good health (with film "Come Clean.")
Major L. G. Mitchell, U. S. A., Washington.

Oral Hygiene and its relation to all health educational activities.
Homer C. Brown, D. D. S., Ex-President, National Dental Ass'n, Columbus.

How can school children be taught preventive dentistry?
Eudore Dubeau, L. D. S., Ecole de Clirungie Dentaire de Montreal.

Children's work in the Rochester Dental Dispensary and in the Schools of Monroe County.
Harvey J. Burkhart, D. D. S., Rochester Dental Dispensary, Director.

Junior Red Cross and School Dental Ambulance Service in rural communities in Nassau County.
Walter J. Multer, Supervising Principal Roslyn High School and Chairman Junior Red Cross Health Activities, Roslyn, L. I.

AN APPEAL TO DENTISTS

For Cooperation in the Fight Against Venereal Diseases.

The war turned the spotlight on many things heretofore neglected or avoided. No disclosures were more startling than those showing the destructive inroads of venereal disease on the health and efficiency of the Army and Navy.

From the time the United States entered the war in April, 1917, to September, 1918, the loss to the Army from venereal disease represented 2,295,000 days of service.

Now the war is over!

The Nation is on its way to a peace basis.

Interest begins to turn from the fighting efficiency of the Army to the reconstructive power of industry; and as it turns this striking fact stands out: **All venereal diseases in the Army were caused by conditions in civilian life. The Army and Navy, as organizations, do not tolerate prostitution.**

Testimony of the Army.

Immediately following the declaration of war the Army Medical Department organized to cope with venereal disease, and one of the first points emphasized by its Surgeon General was that each individual case must be treated under competent medical supervision until cured. He laid special emphasis on two points:

1. The ineffectiveness of self-treatment by the use of simple or patent remedies.

2. The danger of quack doctors, who advertise to treat so-called private diseases.

Nineteen months of war have shown conclusively the value of proper methods of treating venereal cases in the Army.

Maintain Industrial Efficiency.

For the protection of the fighting men as they return home, and to maintain maximum industrial efficiency, venereal disease among the civilian population must be kept under control. There is the same necessity for proper methods of treatment as existed in the service. **Self-treatment and quackery must go.**

Responsibility of Dentists.

Dentists must share the responsibility for preventing the spread of syphilis. It is found in all walks of life, all classes of society, and it is too often not recognized when examinations of the mouth are made for dental treatment. Persons who have lesions in the mouth and know they have syphilis, and admit it, do not as a rule seek the services of a dentist unless

compelled to do so. Those who have lesions and are unaware of their condition present themselves to the dentist unsuspectingly, and for that reason are a serious menace to the health of the dentist and his clientele.

The ease with which the disease is transmitted is well known to the members of the profession. A break in the skin or mucous membrane, a spirochete gaining entrance and passing into the circulation— and infection occurs. Syphilis is no respecter of persons; neither is it a respecter of tissues. The simple scaling of the teeth may, and usually does, involve a breaking in the continuity of gum tissue and exposes the patient to any infection carried on an unclean instrument. In view of the prevalence of syphilis and the difficulty with which the germ of this disease is destroyed, it is of the highest importance that dentists continue to study and practice the principles of asepsis in all phases of their work. It should be borne in mind that the saliva of those infected with syphilis is usually loaded with spirochetes, and that it is not necessary that blood be drawn from the patient in order to infect another thru the application of instruments.

Recognizing the various lesions found in the mouth, which are signs of definite diseases, requires clinical knowledge and experience. Many infectious diseases produce lesions in the mouth, and the study of these lesions, which might enable the dentist to recognize such infection as well as the infection of syphilis, is extremely interesting and should be of material benefit to the dentist. It is believed that if the question of venereal diseases were discussed more freely and frequently in study clubs and dental societies, it would be a benefit to society and reflect credit on the dental profession.

Recent investigations have clearly proved that dental structures are an important part of the human economy. The man who treats his case symptomatically is most likely to fail. Dentists have been confronted with increased responsibility in the matter of general health, not merely because they have aspired to greater things, but because it has been thrust upon them, and the proposition must be met face to face.

Cooperation of Physicians, Druggists, and Advertising Media.

The campaign now being carried on among dentists is similar to those carried on with physicians, druggists, and advertising media. It should interest you to know that approximately 61,000, or nearly 50 per cent, of the physicians of the United States, and that approximately 28,000, or nearly 60 per cent, of the druggists of the United States, have signed and returned agreement cards obligating themselves to cooperate; and in addition to these, more than 99 per cent of all newspapers and periodicals in the United States carrying advertising have pledged themselves not to carry quack advertising

Weigh These Questions.

Will the dental profession accept its share of the burden which this work requires? On receipt of the inclosed franked card, properly signed, the Public Health Service will ask your State Board of Health to supply you with additional scientific and miscellaneous literature.

RUPERT BLUE,
Surgeon General, United States
Public Health Service.

MICHIGAN STATE DENTAL SOCIETY.

The next meeting of the Michigan State Dental Society will be held at the Hotel Statler, Detroit, Mich., April 12, 13 and 14, 1920. The chairman of local arrangements is William H. Waller, 613 Washington Arcade, Detroit.

CLAUDE S. LARNED, Secretary,
614 Post Bldg., Battle Creek, Mich.

MISSOURI STATE DENTAL SOCIETY.

Meeting of the Missouri State Dental Association to be held at the Baltimore Hotel, Kansas City, Missouri, April 12, 13, 14, 1920.

H. C. POLLOCK, Secretary,
724 Metropolitan Bldg.,
St. Louis, Mo.

SOUTHWESTERN DENTAL SOCIETY.

The third annual meeting of the Southwestern Dental Society will be held at El Paso, Texas, on Thursday, Friday and Saturday, March 4, 5 and 6, 1920. A cordial invitation is extended to practitioners of dentistry.

RAYMOND H. GUDGER, Secretary,
Silver City, New Mexico.

SIGMA EPSILON DELTA.

The next regular meeting of the Grand Council of the Sigma Epsilon Delta Fraternity, will be held at the Hotel Astor, 44th Street and Broadway, New York City, on Tuesday, March 23rd, at 8:30 o'clock. Full representation from all chapters is requested.

By order of the Grand Council.

ROBERT E. ELSTER, Master.

KENTUCKY STATE DENTAL SOCIETY.

The next annual meeting of the Kentucky State Dental Association will be held in Louisville, April 7-8-9, 1920.

A program of unusual interest has been planned. Address all correspondence to W. M. Randall, Secy., 1035 Second St., Louisville, Ky.

RECENT PATENTS OF INTEREST TO DENTISTS.

1318254, Dental preparation and making the same, Viggo V. J. Andresen, Copenhagen, Denmark.

1318266, Anesthetic dispenser, Harlon A. Clifford and J. W. Cobb, Greenfield, Mass.

1317980, Dental appliance, Ira Lee, Seattle, Wash.

1317886, Preparing amalgam for dentists' use, Wm. M. Muchow, Evanston, Ill.

1317832, Artificial tooth, Vernon Spencer, Mosman, New South Wales, Australia.

53903, Design, Amalgam remover, Frank L. Grier, Milford, Del.

53904, Design, Alloy-scoop, Frank L. Grier, Milford, Del.

53955, Design, Dental plastic instrument, Daniel A. Zurbrigg, Milford, Del.

53956, Design, Dental carving instrument, Daniel A. Zurbrigg, Milford, Del.

53957, Design, Dental burnisher, Daniel A. Zurbrigg, Milford, Del.

53958 Design, Dental trimming file, Daniel A. Zurbrigg, Milford, Del.

1318522, Tooth-brush, Solomon Alexander, Long Beach, Cal.

1318351, Moistening attachment for dental handpieces, Ira A. Burnett, Chicago, Ill.

1318964, Tooth-brush, Benjamin Chess, New York, N. Y.

1318647, Rotary tooth-brush, Guy B. Collier, Kinderhook, N. Y.

1318751, Therapeutic lamp, John J. Heitz, Chicago, Ill.

1318757, Lighting fixture, Paul M. Hotchkin, Chicago, Ill.

1318783, Combined direct and indirect lighting fixtures, Earle C. McKinnie, Chicago, Ill.

1318586, Hypodermic syringe needle and base, Frank L. Platt, C. N. Hein, and R. R. Impey, San Francisco, Cal.

1318428, Combined tooth-brush holder and tumbler support, Frank E. J. Young, Chicago, Ill.

1319243, Hypodermic syringe, George G. Powers, Los Angeles, Cal.

1320133, Anchorage for dental bridges and plates, Steele F. Gilmore, Indianapolis, Ind.

1320337, Artificial tooth, Simon Myerson, Cambridge, Mass.

1320275, Device for extruding paste from tubes, Edwin G. Roach, New York, N. Y.

1319904, Combined mouth-gag and tongue depressor, Edwin L. Roberts, Nashville, Tenn.

1319737, Dental device and denture construction, Frank M. Wadsworth, Minneapolis, Minn.

1320324, Apparatus for casting dental inlays, Elpha E. Bailey, and J. H. Jacobs, Denver, Colo.

1320536, Automatic injection syringe, Raymond Di Falco and J. De Paul, Ellwood City, Pa.

1320616, Rotary tooth-brush, George S. Hampe, Alhambra, Cal.

1320583, Stand for dental articulators, Wm. M. Stanbrough, Newburgh, N. Y.

1321451, Multiple-jaw rubber-dam clamp, James W. Ivory, Philadelphia, Pa.

1321452, Rubber-dam punch, James W. Ivory, Philadelphia, Pa.

1321465, Dental film holder, Rector P. MacLagan, Park Ridge, Ill.

1321137, Facial contour restorer, Francis M. Morgan, San Francisco, Cal.

1321124, Restoring teeth, Albert D. Pentz, Jr., New York, N. Y.

1321126, Anchor for artificial teeth, George W. Ramsey, Washington, D. C., and G. E. Fritz, Chicago, Ill.

1321129, Dental drill, Frank F. Schlueter, Watertown, Wis.

1321130, Dental drill, Frank F. Schlueter, Watertown, Wis.

1322413, Fountain tooth-brush, Jacob H. Ekmekjian, Pasadena, Cal.

13222424, Forming metal backs for artificial teeth, Ernest Garden, Los Angeles, Cal.

1322355, Denture attachment and means for applying same, Herman Schreier, Sheboygan, Wis.

1322385, Dental articulator, Walter J. Wilson, Petersburg, Ind.

1322387, Motor-driven dental articulator, Walter J. Wilson, Petersburg, Ind.

1322994, Orthodontic appliance, Edward H. Angle, Pasadena, Cal.

1323042, Tooth-brush, Joseph A. Gardner, Memphis, Tenn.

1323161, Method and apparatus for administering anesthetics, Walter V. Goodfellow, Los Angeles, Cal.

1323037, Tooth-brush, Minor T. Holliday, Bozeman, Mont.

1323141, Orthodontia appliance, Jacob L. Young, New Rochelle, N. Y.

Copies of above patents may be obtained for twenty-five cents each, by addressing John A. Saul, solicitor of Patents, Fendall Building, Washington, D. C.

DENTAL CORPS ORDERS FROM WAR DEPARTMENT.

DENTAL CORPS.

Maj. Neal A. Harper to Governors Island, N. Y., for duty.

Capt. Arthur T. Burchill to Montgomery, Ala., aviation repair depot, for duty.

First Lieut. Harold S. Whitney to Carlisle, Pa., general hospital 31, for duty.

—Army and Navy Register, December 6.

The following honorary discharged is colonels only: Cols. Samuel H. Leslie and Frank P. Stone.

Lieut. Col. E. Henry Valentine to Camp Jackson, S. C., for duty.

The following honorably discharged as lieutenant colonels only: Lieut. Cols. Lee S. Fountain, E. Henry Valentine and Charles M. Taylor.

Maj. John L. Schock and Capt. Nathan C. Pickles to San Francisco and to Manila, P. I., about Jan. 5, for duty.

Majs. Thomas C. Daniels and James L. P. Irwin from duty in Philippine Department to United States and report for instructions.

The following honorary discharged as colonels only: Majs. Leslie D. Baskin and William C. Webb, Jr.

Capt. Waldo J. Adams to Whipple Barracks, Ariz., general hospital 20, for duty.

Capt. Merle W. Catterlin to Fort Sheridan, Ill., general hospital 28, for duty.

Capt. Julius Link Bishof to Denver, Colo., general hospital 21, for duty.

Capt. Solomon J. Z. Gantz to Camp Lewis, Wash., for duty.

Capt. Leroy T. Asche to Governors Island, N. Y., for duty.

—Army and Navy Register, December 13.

Capt. Warren C. Caldwell report by wire to commanding general Southern Department, for duty.

Capt. Alexander M. Smith, Jr., to Fort Bliss, Tex., base hospital for duty.

—Army and Navy Register, December 20.

Maj. Benjamin C. Warfield, report by wire to commanding general Northeastern Department for assignment.

First Lieut. Arthur F. Bokman, to Columbus barracks, Ohio, for duty.

—Army and Navy Register, December 27.

Lieut. Col. George I. Gunckel (promoted subject to examination), having been examined for promotion by a board of officers, and found physically disqualified for the duties of a lieutenant colonel in dental corps, by reason of disability incident to the service, his retirement from active service as a lieutenant colonel is announced; proceed home.

The following to San Francisco and to Vladivostok, Siberia, Jan. 5, for duty; Lieut. Col. John R. Ames, Capts. Claude B. Hollister, Edward C. Alley, First Lieuts. George M. Krough and Chester B. Parkinson.

Maj. Frederick R. Wunderlich from duty with American expeditionary forces, Vladivostok, Siberia, to United States and report for instructions.

Capts. Daniel Bratton, Oscar W. Thompson to Camp Zachary Taylor, Ky., for duty.

First Lieut. Henry A. Winslow to general hospital 2, Fort McHenry, Md., for duty.

—Army and Navy Register, January 3.

Maj. Charles M. Taylor to Walter Reed General Hospital, D. C., for duty.

Maj. William D. White to Alcatraz, Calif., disciplinary barracks for duty.

Maj. Earle J. McClung report by wire to commanding general Western Department for assignment to duty.

Maj. Robert R. Luce, honorably discharged.

Capt. Joseph L. Rahm to Arcadia, Calif., Ross Field, for duty.

Capt. John L. Davis to Camp Upton, N. Y., for duty.

Capt. Leon M. Muedeking to Fort Sheridan, Ill., general hospital 28, for duty.

First Lieut. Herbert E. Guthrie to San Antonio, Tex., Kelly Field for duty.

—Army and Navy Register, January 10.

The resignation of Maj. E. Henry Valentine is accepted.

Maj. David Archibald Proctor and First Lieuts. George Robson White and William Alfonso Baker are honorably discharged.

Capt. Arthur Clay Foard to Walter Reed General Hospital, Washington, for duty.

First Lieut. Henry Lake Gilmour, Jr., to Camp A. A. Humphreys, Va., for duty.

Appointment of Clark Wayne Russell as first lieutenant, dental corps, Regular Army, from Oct. 28, 1919, is announced; to Fort Logan, Colo., for duty.

Appointment of Joseph Aloysius Murray as first lieutenant, dental corps, Reguar Army, from Oct. 30, 1919, is announced; to Camp Dix, N. J., for duty.

Appointment of Forest Vernon Bockey as first lieutenant, dental corps, Regular Army, from Oct. 30, 1919, is announced; to report to commanding general, American Forces in Siberia, for duty.

Appointment of James Lawrence Olsen as first lieutenant, dental corps, Regular Army, from Oct. 31, 1919, is announced; to Camp Meade, Md., for duty.

Appointment of Alexander MacKenzie Telfer as first lieutenant, dental corps, Regular Army, with rank from Nov. 1, 1919, is announced; to Walter Reed General Hospital, Washington, for duty.

Appointment of Clyde Wakefield Scogin as first lieutenant, dental corps, Regular Army, from Nov. 3, 1919, is announced; to Alcatraz, Calif., disciplinary barracks for duty.

The following temporary officers of the dental corps have been appointed first lieutenants, dental corps, Regular Army: Capt. Page Purnell Albert Chesster, Oct. 25, 1919; First Lieut. Ralph Eugene Morgan, Oct. 26, 1919; First Lieut. Joseph Hayden Jones, Oct. 29, 1919; First Lieut. Glenn Dale Lacey, Oct. 31, 1919; First Lieut. Harold Snell Whitney, Nov. 1, 1919; First Lieut. Egbert Wesley van Delden Cown, Nov. 2, 1919; Capt. Beverley Morrison, Epes, Nov. 4, 1919; Capt. Warren Charles Caldwell, Nov. 4, 1919.

—Army and Navy Register, January 17.

Maj. Oscar P. Snyder to Camp Grant, Ill., for duty.

Maj. Rex McK. McDowell to Washington and report to director of purchase and storage for duty.

Maj. Nathaniel Barnard to Jefferson Barracks, Mo., for duty.

The resignation by Homer L. Sams, dental corps, as temporary captain and as first lieutenant (Regular Army) accepted.

1st Lieut. Clyde W. Scogin to Denver, Colo., general hospital 21, for duty.

1st Lieut. Robert C. Craven to Camp Grant, Ill., for duty.

1st Lieut. Earl G. Gebhardt to Camp Grant, Ill., for duty.

—Army and Navy Register, January 24.

DENTAL CORPS.

Resignation by Maj. Ben H. Sherrard accepted.

Capt. Thomas M. Page, to Camp Jackson, S. C., for duty.

1st Lieut. Clyde W. Scogin, to Fort McDowell, Calif., for duty.

1st Lieut. Frank R. Gould, honorably discharged.

—Army and Navy Register, January 31.

In Memoriam

HENRY WILLIAM MORGAN, M. D., D. D. S.

Died, at his home in Nashville, Tenn., on January 17th, 1920, in the 67th year of his age, Dr. Henry W. Morgan.

Dr. Morgan was born October 25th, 1853. He was the son of Dr. Wm. H. and Sarah Noel Morgan. He was educated in the Nashville public and high schools. He graduated in medicine in Vanderbilt University and was graduated in 1873, being the first man to receive a degree from that institution. In 1875 he was graduated from the Philadelphia Dental College as D. D. S.

In 1876 he began the practice of dentistry in his father's office in Nashville, and continued till forced to retire by ill health less than a year ago. The following is taken from the Nashville Banner:

"Dr. Morgan became professor of operative dentistry in the Vanderbilt School of Dentistry in 1886, and from that time until last June was connected with Vanderbilt in such capacity. In 1911 he was elected dean of the school of dentistry, a position which his father, who had founded the school, had held before him. He was connected with the university in this capacity until June, 1919, when failing health caused him to decline re-election.

He had held various offices in both state and national dental associations. He was ex-president of the old Southern Branch of the National Dental Association, ex-president of the National Association of Dental Teachers. He held the same office in both the Tennessee Dental Association and Nashville Dental Association.

During the war he served on the Dental Reserve branch of the Council of National Defense as well as chairman of the Ad Interim committee which was an intermediary between the War Department and Dental Reserve. He was also ex-chairman of the Foreign Relation committee of the National Association of Dental Faculties.

Dr. Morgan was also ex-president of the Vanderbilt Alumni Association. At the time of his death he was president of the board of trustees of Walden University and Meharry Medical College, a position his father had held before him.

He was a member of the West End Methodist Episcopal Church, serving on the board of trustees of the church for years. He took an active part in the affairs of his denomination.

He was also affiliated with a number of fraternal organizations during his life. He was a 33rd degree honorary Scottish Rite Mason, which honor was conferred upon him by the supreme council at Washington, D. C., in 1917. He was a member of Cumberland Lodge, No. 8, F. & A. M. and of the Scottish Rite bodies of the Valley of Nashville. He was past grand chancellor of the Knights of Pythias of Tennessee and past supreme representative and past grand regent of the

Royal Arcanum, and in 1917-18 was the supreme grand master of the Delta Sigma Delta dental fraternity.

He was identified with numerous local clubs and organizations, being a charter member of the Kiwanis Club, a member of the Commercial Club and of the old Nashville Board of Trade. He belonged to the Old Oak Club and the Round Table Club. He served as chairman of the old committee of one hundred in its fight for cleaner city politics.

He was also a member of the executive committee of the board of trustees of the Monteagle Sunday School Assembly for a number of years.

Dr. Morgan was married to Miss Matilda Alloway Evans of Nashville November 3, 1880. Mrs. Morgan and the five children of the marriage, William H. Morgan, of Dallas, Tex., Mrs. William C. Weaver, Mrs. Palmer C. McClester, Mrs. S. Cecil Ewing and Dr. Walter M. Morgan of Nashville, survive him. His two brothers, Jo B. Morgan and Garnett N. Morgan, live in Nashville, while his sister, Mrs. Chas. H. Noyes, lives in Warren, Pa."

Thus are chronicled in brief array the chief events in the life and work of this splendid man. To those who knew him well the record is all too meagre. It does not tell of the many sterling attributes of character which signalized his every act in his intercourse with his fellow man. It does not tell of his charm of manner, his courtliness of demeanor, his soundness of sentiment, his sterling character, his pronounced principles, or his superb and supreme manhood.

Dr. Morgan was endowed as are few men with those rare qualities of mind and heart which stamped him at once as a true son of the South. He left an impress not only on the friends with whom he came in daily contact, but on the world-at-large, as an example of the highest type of professional gentleman. His kindliness, his geniality, his unselfishness and his dignity of bearing all endeared him to his fellowmen, and commanded their constant respect and admiration.

The world is better because of the life of Henry Morgan; the profession is richer because he graced its ranks; and his immediate family and friends are consecrated and hallowed because he loved and labored among them. His life was an inspiration, and his memory is a blessed benediction—an influence extending for good thru all the vista of the coming years. C. N. J.

RESOLUTIONS.

Dr. Henry William Morgan.—God, the All-powerful Ruler of the destinies of man, who works in a wondrous way, his mysteries to perform, has but now taken from us and unto Himself our greatly beloved brother and friend, Dr. Henry William Morgan. A splendid christian gentleman, loyal friend, true to his ideals of our profession, outspoken, fearless, yet an inspiration to all who knew him, he was a man among men, revered and loved.

He served the profession of dentistry for over forty years of his life well and intelligently. For many years he gave largely of his time and ability to the advancement of his profession, to the better opportunities and facilities for teaching students the advanced thoughts and sciences of the day.

As professor and instructor in the School of Dentistry, of Vanderbilt University, he stood in the front rank of the highest class of educators. There is not a dentist or a dental organization anywhere that has not suffered an irreparable loss in his death.

He was a contemporary of Black, McKellops, Bonwill, Noyes, Cushing and Johnson. He ran an even race with these eminent men in high professional attainments.

We are confident that the spirit of his energies and love of fellowman still

lives and will rest among us for all time and will forever contribute to our better welfare professionally and individually.

May his soul rest in peace and may the memory of him and his deeds be graven deep in our hearts as well as on the tablets of the Hall of Fame erected in honor of the great men of our profession.

We, the Society of Dental Surgeons of Nashville, Tenn., do feel our loss in the taking away of our beloved member more deeply than we know how to express.

A great sympathy goes out to the family. We mourn with them his passing from us, but glory in the knowledge that he rests in the bosom of his God.

D. M. CATTELL,
J. H. PHILLIPS,
R. BOYD, BOGLE,
Committee.

RESOLUTIONS.

Dr. B. Holly Smith.—At a meeting of the Board of governors of the Maryland State Dental Association held January 23, 1920, the following resolutions were adopted:

Whereas: We, members of the Maryland State Dental Association, have heard with sincere sorrow of the sudden passing of our friend and fellow member, Dr. B. Holly Smith, and

Whereas, we desire to express our love for and manifest our appreciation of so valuable a representative of dental science. Therefore, be it

Resolved, that in the death of Dr. Smith, we have lost a most estimable friend and the profession, locally and nationally, an earnest, energetic and worthy advocate of the highest ideals in the advanced methods of alleviating human suffering from oral defects and who by his untiring efforts has succeeded in establishing free dental clinics in the public schools of Baltimore. Be it further

Resolved, that by wise counsel and matured advice, his pleasing presence will be sadly missed in the meetings of his state, and national conventions to which he was most loyal and devoted, manifesting keen interest in every detail, that would aid the progress of his chosen profession, and wherein his wisdom and understanding of dental science made impressions that were signally felt. Be it further

Resolved, that the good fellowship and enduring friendship that his winsome presence made, wherever he went, will always remain a delightful memory to those fortunate enough to have made contact with so vital and energizing a spirit. Be it further

Resolved, that we have spread on the records of this association these resolutions, that a copy be presented to his family and also to the various dental journals of the country and a copy be printed in the Daily Press, of Baltimore.

W. W. DUNBRACCO,
B. LUCIEN BRUN,
Committee.